

SLEEP MEDICINE CLINICAL SERIES

BRPT - Advanced Titration Certificate

RPSGT(ATC) Certified - 2026

ADVANCED TITRATION

A Comprehensive Clinical Reference

EXAM COMPOSITION

75 multiple-choice questions - 3 domain areas - 70% to pass

DOMAIN 1 - 20%	DOMAIN 2 - 30%	DOMAIN 3 - 50%
Advanced Knowledge • Respiration & ventilation • Gas exchange - Respiratory drive • Breathing patterns • EtCO₂ / TcCO₂ / SpO₂ / ABG	Pathophysiology & Clinical Presentation • Sleep-related breathing disorders • OSA (adult / pediatric / infant) • Central sleep apnea (six adult subtypes per ICSD-3-TR) • Hypoventilation (OHS / CCHS / ROHHAD / NMD)	Evaluation & Treatment • PAP therapy - CPAP / APAP / Bilevel • ASV - Volume-targeted ventilation • Oxygen, oral appliance, neurostimulators • HFNC, surgical, behavioral therapy

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Advanced Titration

A Comprehensive Clinical Reference

(revised)

for the BRPT Advanced Titration Certificate

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FIRST EDITION (revised)

2026

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Advanced Titration: A Comprehensive Clinical Reference for the BRPT Advanced Titration Certificate

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Audience

Audience	Description
Primary (exam candidates)	Active RPSGT (Registered Polysomnographic Technologist) and CCSH (Certified in Clinical Sleep Health) credential holders preparing for the BRPT Advanced Titration Certificate (ATC) examination, as these are the only two credentials eligible to sit for the ATC exam.
Reference users	Respiratory therapists (RT, RRT, RTRP, RCP); sleep medicine physicians and fellows; pulmonologists and critical care clinicians using noninvasive ventilation; polysomnography and respiratory therapy students; sleep laboratory nurses and managers; sleep technology educators.

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The information presented in this book is intended for use by credentialed healthcare professionals, students, and candidates preparing for the Board of Registered Polysomnographic Technologists (BRPT) Advanced Titration Certificate (ATC) examination (18). The author is a credentialed sleep technologist and respiratory therapist (see Author Credentials on the Publication page). The author is not a licensed physician, and nothing in this work constitutes the practice of medicine.

Clinical recommendations, titration thresholds, pressure parameters, device selection criteria, and treatment algorithms summarized in this work are derived from published guidelines current at the time of writing, principally those of the American Academy of Sleep Medicine (AASM). Medicine is a rapidly evolving discipline. Guidelines change, new evidence emerges, and individual patient circumstances vary. Readers must verify all clinical information against the most current published guidelines, the prescribing physician's orders, their institutional protocols, and the device manufacturer's instructions for use before applying any concept discussed in this work to actual patient care.

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Preface

When the Board of Registered Polysomnographic Technologists introduced the Advanced Titration Certificate (ATC) examination (18), many of us preparing for it found ourselves stitching together fragments from disparate sources: a section from a textbook, a guideline document, a manufacturer's clinical bulletin, lecture notes from a past course, snippets of forum discussion. The breadth of the examination, spanning gas-exchange physiology, central and obstructive sleep apnea, hypoventilation syndromes, the full complement of positive-airway-pressure modalities, oxygen therapy, oral appliances, neurostimulators, surgical considerations, and behavioral interventions, made a single, consolidated reference feel necessary. This book is the resource I needed and could not find.

Who this book is for. This book is designed first and foremost as exam-preparation material for the only two BRPT credential holders eligible to sit for the Advanced Titration Certificate (ATC) examination: the **Registered Polysomnographic Technologist (RPSGT)** (19) and the **Certified in Clinical Sleep Health (CCSH)**. If you do not hold an active RPSGT or CCSH credential, you cannot register for the ATC examination at this time, but you can still use this book as a clinical reference.

The content is also useful as a working reference for any practitioner whose role involves manual PAP titration, complex sleep therapy, or noninvasive ventilation: **respiratory therapists in sleep laboratories or NIV programs** (RT, RRT, RTRP, RCP), **sleep medicine fellows and attending physicians, pulmonologists and critical care clinicians** using bilevel and adaptive modalities, **polysomnography and respiratory therapy students, sleep laboratory nurses and managers**, and **educators** teaching titration in formal training programs. The depth of coverage assumes baseline familiarity with sleep architecture, scoring rules, and standard polysomnographic montage; readers entering sleep medicine from an adjacent field may benefit from pairing this book with an introductory text (16).

How the book is organized. Content follows the BRPT ATC examination blueprint. Chapter 1 covers Domain 1 (Advanced Knowledge, 20 percent of the exam): respiratory physiology, ventilatory control, breathing patterns, and bedside monitoring. Chapter 2 covers Domain 2 (Pathophysiology and Clinical Presentation, 30 percent): obstructive sleep apnea, the central sleep apnea subtypes recognized by the ICSD-3-TR (2), and the hypoventilation syndromes. Chapters 3 and 4 cover Domain 3 (Evaluation and Treatment, 50 percent): the AASM titration guidelines themselves (11), every PAP modality, oxygen therapy, oral appliance therapy, the three neurostimulator devices currently in use, alternative treatments, and pediatric titration. Chapter 5 is an illustrated waveform atlas. Chapter 6 is a quick-reference summary plus a section on exam strategy, drawn from analysis of common missed-question patterns. Chapter 7 lists the primary references in Vancouver order of first appearance, followed by an alphabetical index.

How to use this book. Read Chapter 1 carefully, even slowly. The physiology is the foundation; nothing in the later chapters makes sense without it. Then move through Chapters 2 through 4 in order. Use Chapter 5, the waveform atlas, alongside the chapters as you encounter each pattern or device. Save Chapter 6 for the final week before your examination, when you need to consolidate the key numbers and rules. The reference list in Chapter 7 is the ground truth: when in doubt, the original guidelines outrank any study guide, including this one.

Citation style. In-text citations follow the Vancouver numeric sequence style: each reference is assigned a number in order of first appearance in the text, and the number is inserted in parentheses at the relevant point.

The full reference list appears in Chapter 7 in the same numeric order, allowing readers to follow any in-text citation directly to its source. A comprehensive alphabetical index follows the reference list.

A note on currency. The content reflects published guidelines as of early 2026. Sleep medicine is a moving field; the AASM published an updated central sleep apnea treatment guideline in late 2025 (5) that softens the historical SERVE-HF contraindication for adaptive servo-ventilation (3), and the field continues to evolve. Where the published guideline is itself the testable item, this book follows the guideline. Where exam content lags clinical practice, this book notes both frameworks. Always consult the most current source.

If this book helps even one technologist walk into the ATC examination feeling that the material is approachable rather than overwhelming, the long nights spent compiling it will have been worth it. Best of luck on your examination, and more importantly, thank you for the work you do for patients with sleep-disordered breathing. Your skill at the bedside changes lives.

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California, 2026

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Revision Notice — First Edition, May 2026

This document has been revised from its initial release to correct several factual inaccuracies identified during post-publication review. The corrections affect specific clinical thresholds, FDA device-labeling history, and one titration-algorithm statement, but do not alter the overall scope, structure, or pedagogical approach of the work.

Corrections applied:

1. Section 3.2 — Bilevel PAP titration algorithm. The directionality of the pressure-adjustment rule was corrected. Residual obstructive apneas require an increase in both IPAP and EPAP; residual hypopneas, RERAs, or snoring require an increase in IPAP only.
2. Section 3.5 and Section 6.5 — Hypoglossal nerve stimulator (Inspire®) FDA labeling history. The age, AHI, and BMI indications were restated to reflect the layered FDA approval history: primary indication age ≥ 22 (PMA P130008, 2014); expanded indication for ages 18 – 21 (P130008/S039, April 2020); indication for ages 13 – 18 with Down syndrome and severe OSA (P130008/S089, March 2023); AHI and BMI thresholds expanded to ≤ 100 and ≤ 40 respectively (P130008/S090, June 2023).
3. Section 4.2 — Pediatric PAP titration pressure caps. The maximum pressures for children aged 12 years and older were corrected to align with the Kushida 2008 guideline: CPAP maximum 20 cmH₂O and BPAP IPAP maximum 30 cmH₂O. Children under 12 retain the lower caps (15 and 20 cmH₂O).
4. Section 2.1 and Chapter 6 — Pediatric OSA diagnostic threshold. The diagnostic criterion was corrected to obstructive AHI ≥ 1 event per hour per ICSD-3-TR and the AASM Scoring Manual. The ≥ 1.5 figure, which appears in some research literature and clinical conventions, is noted but is not the formal AASM diagnostic threshold.
5. Section 3.5 — eXciteOSA® maintenance schedule. The maintenance frequency was corrected to once weekly per the FDA-cleared labeling (DEN200018). The adverse-event list was also expanded to match the FDA De Novo decision summary.
6. Section 2.1 — Adult OSA diagnostic criterion. The text was clarified to reflect that the ICSD-3-TR criterion is formally based on the obstructive respiratory disturbance index (RDI), which includes RERAs in addition to apneas and hypopneas, while noting that AHI and RDI are often used interchangeably in clinical practice.

No changes were made to the reference list, the citation numbering, the chapter structure, or the page layout beyond what was necessary to accommodate the corrections above. Readers who downloaded the original release are encouraged to replace it with this revised version.

The author thanks the colleagues and readers whose careful review identified these issues. Any remaining errors are the author's own, and corrections or comments are welcome via the author profiles listed in the Preface.

Samantha Faye O. Tina
California, May 2026

Acknowledgments

No book is the work of a single author, even one bearing a single name on its cover. I owe a debt to the clinicians, researchers, and guideline-writing task forces whose published work forms the intellectual backbone of this reference, in particular the authors of the AASM Clinical Guidelines for the Manual Titration of Positive Airway Pressure (Kushida and colleagues, 2008) (11), the AASM Scoring Manual editorial board (6), and the many investigators whose pivotal trials (STAR (12), SERVE-HF (3), CANPAP (4), Costanzo (13), and others) are cited throughout.

I am grateful to my colleagues in the polysomnography and respiratory therapy community, whose questions, corrections, and clinical wisdom shaped what is and is not included in these pages. To my mentors in sleep medicine: thank you for showing me what good titration looks like at three in the morning when nothing in the textbook seems to apply.

Finally, to my family, whose patience with the long evenings and weekends of writing made this work possible: this book is for you as much as for the technologists who will read it.

S.F.O.T.

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Chapter 1

Advanced Knowledge - Domain 1 - Exam Weight 20 percent

Domain 1 of the Board of Registered Polysomnographic Technologists (BRPT) Advanced Titration Certificate examination (18) covers the physiology of respiration and ventilation (15), the monitoring tools used during a polysomnogram (16), and the patterns of breathing that point to specific pathologies. The material in this chapter forms the foundation on which every clinical titration decision in later chapters depends, so it is worth reading slowly and making sure each concept is genuinely understood rather than simply memorized.

1.1 Gas Exchange

Gas exchange takes place across the alveolar-capillary membrane, the extremely thin barrier where each alveolus is wrapped by pulmonary capillaries. Oxygen diffuses from the air inside the alveolus into the blood, while carbon dioxide diffuses in the opposite direction, from the blood into the alveolus where it can be exhaled. This entire process is passive, meaning it requires no energy from the body, because both gases move from areas of higher partial pressure to areas of lower partial pressure. How well this exchange works depends on four interrelated factors.

Ventilation-to-Perfusion Matching (the V/Q ratio)

For gas exchange to be efficient, every alveolus that receives fresh air must also be sitting next to a capillary that delivers blood. The ratio of alveolar ventilation to pulmonary blood flow, abbreviated as the V over Q ratio, expresses how well this match is maintained. The ideal V/Q ratio in a healthy lung is approximately 0.8. When the ratio falls below 0.8, blood is reaching alveoli that are not being ventilated properly, and the result is essentially the same as if that blood had bypassed the lungs altogether. This is called a shunt effect and it produces hypoxemia, which means low blood oxygen. When the V/Q ratio rises above 0.8, the opposite happens: air is reaching alveoli that are not receiving blood, so the ventilation is being wasted on areas where no gas exchange can occur. This is called a dead-space effect and it is characteristic of conditions such as pulmonary embolism, where a clot blocks blood flow to a portion of the lung.

Diffusion Capacity

The diffusion capacity of the lung for carbon monoxide, abbreviated DLCO, is a clinical test that measures how easily gas crosses the alveolar-capillary membrane. The number is reduced in diseases that either destroy alveolar surface area, such as emphysema, or thicken the membrane itself, such as pulmonary fibrosis. It is also reduced in anemia, because there are fewer red blood cells to carry the gas across. Diffusion capacity rises in conditions that increase blood volume in the lung, such as polycythemia (an abnormally high red blood cell count) and during exercise.

Dead Space

Not every breath of air actually participates in gas exchange. The first portion of every inhalation simply fills the conducting airways (the trachea and bronchi) and never reaches an alveolus. This volume, approximately 150 milliliters in an average adult, is called **anatomic dead space**. It tends to increase with age and with airway diseases such as chronic bronchitis. In addition to anatomic dead space, there is also **alveolar dead space**, which refers to alveoli that are ventilated but receive no blood flow (for example during a pulmonary embolism).

or in shock states where perfusion fails). When the two are added together, the total is called **physiologic dead space**, and it can be estimated using the Bohr equation: the ratio of dead-space volume to tidal volume equals $(\text{PaCO}_2 \text{ minus } \text{PeCO}_2) \text{ divided by } \text{PaCO}_2$, where PeCO_2 is the mixed expired CO_2 .

Key Equations - Gas Exchange

Alveolar Gas Equation: $\text{P}_\text{A}\text{O}_2 = \text{FiO}_2 \times (\text{P}_\text{atm} - \text{P}_\text{H}_2\text{O}) - (\text{PaCO}_2 \div \text{RQ})$

Normal arterial values: PaO_2 80 to 100 mmHg, PaCO_2 35 to 45 mmHg, SaO_2 at least 95 percent, pH 7.35 to 7.45.

Alveolar-arterial (A-a) gradient on room air: $\text{P}_\text{A}\text{O}_2$ minus PaO_2 ; normal is less than 15 mmHg. The gradient rises with V/Q mismatch, true shunt, or impaired diffusion.

Respiratory quotient (RQ): the ratio of CO_2 produced to O_2 consumed by cellular metabolism. It is approximately 0.8 on a mixed diet, 1.0 on a pure carbohydrate diet, and 0.7 on a pure fat diet.

1.2 Respiratory Drive

Breathing happens automatically because the brainstem contains specialized groups of neurons that generate a rhythmic pattern of neural impulses and send them down to the diaphragm and other respiratory muscles (15). These impulses are modulated continuously by feedback from chemical sensors that monitor the blood. Understanding where each of these structures sits inside the brain, and how they respond to changes in oxygen and carbon dioxide, is essential for understanding both normal breathing and the breathing disorders encountered during polysomnography.

The Central Pattern Generator

The basic rhythm of breathing is generated by a small cluster of neurons called the **pre-Bötzinger complex**, which sits in the medulla oblongata. The medulla is the lowest part of the brainstem, located just above the spinal cord and below the pons. Because the pre-Bötzinger complex fires automatically without any conscious input, breathing continues during sleep and even during unconsciousness, as long as the medulla itself is functioning.

Central Chemoreceptors

The central chemoreceptors are also located in the medulla, very close to the rhythm generator described above. They are surrounded by cerebrospinal fluid (the clear fluid that bathes the brain and spinal cord), and they monitor the pH of this fluid. The pH of the cerebrospinal fluid reflects the level of carbon dioxide in arterial blood, because CO_2 crosses freely from blood into cerebrospinal fluid, where it becomes carbonic acid and lowers the pH. So when arterial CO_2 rises, the cerebrospinal fluid becomes more acidic, and the central chemoreceptors respond by increasing the breathing drive. In healthy people these central chemoreceptors are the primary driver of breathing, accounting for roughly 70 to 80 percent of the minute-to-minute control of ventilation. Their response is relatively slow, taking one to two minutes to fully react to a change in CO_2 .

Peripheral Chemoreceptors

The peripheral chemoreceptors sit outside the brain. The most important ones are the carotid bodies, which are tiny structures located at the bifurcation of each common carotid artery in the neck. Smaller aortic bodies sit along the arch of the aorta. Unlike the central chemoreceptors, which respond mainly to CO_2 , the peripheral chemoreceptors respond strongly to a drop in arterial oxygen, particularly when PaO_2 falls below about 60 mmHg. They also respond to high CO_2 and to acidosis. Their response is fast, taking only about 15 to 20 seconds to fire.

The Hering-Breuer Reflex

Stretch receptors located throughout the lung tissue sense how much the lung has expanded during a breath. When the lung approaches full inflation, these receptors send a signal up the vagus nerve (the tenth cranial nerve) to the brainstem, which then cuts off the inspiratory drive. This is called the Hering-Breuer reflex, and it prevents over-inflation of the lungs.

Hypercapnic Ventilatory Response (HCVR)

The hypercapnic ventilatory response, abbreviated HCVR, describes how much a person's breathing increases in response to a rise in arterial carbon dioxide. In healthy adults the relationship is roughly linear (meaning it forms a straight line on a graph), with minute ventilation rising by about 2 to 3 liters per minute for every 1 mmHg increase in PaCO_2 . To put that in plain language: if a healthy person's PaCO_2 rises by 10 mmHg, that person's total breathing (minute ventilation, which equals tidal volume times respiratory rate) should increase by about 20 to 30 liters per minute. That is a large and clinically noticeable response. The HCVR is blunted, meaning the slope of the response is reduced, in several important conditions, including obesity hypoventilation syndrome (OHS), chronic opioid use, and advanced chronic obstructive pulmonary disease (COPD).

Hypoxic Ventilatory Response (HVR)

The hypoxic ventilatory response, abbreviated HVR, describes how much breathing increases when arterial oxygen falls. Unlike the HCVR, this relationship is not linear: ventilation does not change much at all until PaO_2 drops below about 60 mmHg, at which point ventilation rises sharply. This is one of the reasons that pulse oximetry alone is not a sensitive early warning sign of respiratory failure in patients with an intact central drive. The HVR is also blunted in people who have been chronically hypoxemic, such as those living at high altitude or those with severe chronic lung disease.

CLINICAL PEARL - CO₂ Drive Versus Hypoxic Drive

In healthy individuals, CO₂ is the primary driver of breathing. However, in people who chronically retain CO₂ (such as those with severe COPD, OHS, or congenital central hypoventilation syndrome, abbreviated CCHS), the central chemoreceptors gradually reset to the higher CO₂ level and stop responding to it. In these patients, the hypoxic drive (the response to low oxygen) takes over as the dominant signal that keeps them breathing.

This is why giving high-flow oxygen to a known CO₂ retainer can be dangerous: by raising the oxygen level, you abolish the only remaining stimulus the patient has to breathe, and hypoventilation worsens. The safer approach is to use a Venturi mask or carefully titrated oxygen, with a target SpO₂ of 88 to 92 percent.

Sleep-State Respiratory Physiology

Sleep Onset: Loss of Wakeful Drive. During wakefulness, breathing receives a tonic stimulus from the reticular activating system that is independent of CO₂. This is called the wakefulness drive. At sleep onset, the wakefulness drive is withdrawn, and breathing now depends almost entirely on chemical (CO₂) drive. Three things happen as a result. First, tidal volume falls and respiratory rate drops slightly. Second, the resting PaCO₂ climbs by approximately 3 to 7 mmHg (sleep onset hypoventilation is physiologic, not pathological). Third, the upper-airway dilator muscles (principally the genioglossus) lose their tonic activity, so upper-airway resistance rises. This combination - withdrawal of wakefulness drive, mild hypoventilation, and increased upper-airway resistance - is what unmasks OSA at sleep onset in predisposed patients.

Upper-Airway Collapsibility Is Worst at End-Expiration. On the BRPT examination, you may be asked when during the respiratory cycle the upper airway is most prone to collapse. The answer is at end-expiration. There are two reasons. First, lung volume is at its lowest at end-expiration, and lower lung volumes reduce the caudal traction (tug) that the lungs normally exert on the upper airway, leaving the pharynx more compliant. Second, at end-expiration intraluminal pressure is at its lowest point relative to surrounding extraluminal pressure, so the transmural pressure favors collapse. This is also why EPAP works: EPAP raises the intraluminal pressure at end-expiration and prevents the airway from collapsing before the next inspiration begins.

NREM Sleep: Chemical Drive Dominates. During NREM sleep, especially N3 (slow-wave sleep), ventilation is regular and depends primarily on CO₂. The apneic threshold (the CO₂ level below which the brainstem stops sending the drive signal) is unmasked during NREM, which is why central apneas, periodic breathing, and Cheyne-Stokes patterns are most prominent in N1 and N2.

REM Sleep: The High-Risk Stage for Hypoventilation. REM sleep produces near-total skeletal muscle atonia. The diaphragm is preserved, but the accessory respiratory muscles (intercostals, sternocleidomastoids, scalenes) are essentially paralyzed. As a result, tidal volume in REM depends almost entirely on the diaphragm. In any patient whose diaphragm cannot independently sustain adequate ventilation, hypoventilation appears **FIRST** and **WORST** in REM. This is the physiology behind REM-related hypoxemia in obesity hypoventilation syndrome, neuromuscular disease (especially diaphragm-sparing dystrophies early in their course), and severe COPD. REM also produces the longest and most desaturating obstructive events,

because the loss of upper-airway dilator tone is most pronounced in REM. Chemical responsiveness (HCVR and HVR) is also blunted in REM, so arousal occurs at a higher CO_2 and lower SpO_2 than in NREM.

1.3 Breathing Patterns

Several distinctive breathing patterns are encountered in clinical sleep medicine, and recognizing them on a polysomnogram tracing is an examination essential. Each pattern has a characteristic shape and a typical underlying cause. A full illustrated comparison appears in Chapter 5; the summary below describes each pattern in words.

Pattern	Mechanism or Cause	Key Feature
Eupnea	Normal breathing	Regular rate of 12 to 20 breaths per minute, consistent depth, equal ratio of inspiration to expiration.
Cheyne-Stokes	Congestive heart failure, stroke, renal failure, high altitude	Crescendo-decrescendo pattern in which tidal volume gradually grows, then gradually shrinks, then stops in a central apnea, and the cycle repeats every 45 to 90 seconds.
Biot or Ataxic	Lesion of the medulla (trauma, meningitis, brainstem herniation)	Completely irregular rate AND depth, with sudden unpredictable apneas. No pattern at all.
Apneustic	Lesion of the pons (infarction, demyelinating disease). The pons is the section of brainstem above the medulla.	Prolonged inspiratory HOLD, almost a gasp, followed by a brief expiration.
Kussmaul	Metabolic acidosis (diabetic ketoacidosis, advanced kidney disease)	Deep, regular, rapid sighing breaths that blow off CO_2 to compensate for the acidosis.
Central sleep apnea	Low CO_2 threshold, congestive heart failure, opioid use	Normal breaths interrupted by apneas during which there is no chest or abdominal movement at all (no respiratory effort).

1.4 Monitoring Modalities

Modern polysomnography and PAP titration laboratories use four main tools to monitor ventilation and oxygenation (16): end-tidal carbon dioxide (EtCO_2), transcutaneous carbon dioxide (TcCO_2), pulse oximetry (SpO_2), and arterial blood gas analysis (ABG). Each has a specific use, and each has limitations that the technologist must understand to interpret results correctly.

End-Tidal CO_2 (Capnography)

End-tidal CO_2 is the partial pressure of carbon dioxide in the air a patient exhales at the very end of an expiration. At that point, the air leaving the mouth has come all the way up from the alveoli and reflects the alveolar CO_2 , which is in turn very close to arterial CO_2 . A normal EtCO_2 reading is 35 to 45 mmHg, typically

running about 2 to 5 mmHg lower than the actual PaCO_2 because of dilution by dead-space gas from the conducting airways.

A capnogram is a graph of the CO_2 level in exhaled air plotted against time. It has four recognizable phases. **Phase 0** is the period of inspiration, during which the CO_2 reading is zero because the patient is breathing in fresh air. **Phase I** is the very beginning of expiration, while the air from the conducting airways (the anatomic dead space) is being exhaled; this air has no CO_2 in it, so the tracing stays flat at zero. **Phase II** is a steep upslope, produced as alveolar gas (which has CO_2 in it) begins to mix with the dead-space gas and reach the sensor. **Phase III** is the alveolar plateau, a nearly flat top with a slight upward tilt. The value at the very end of phase III, just before the next inspiration begins, is the EtCO_2 number.

Normal Capnogram (End-Tidal CO_2)

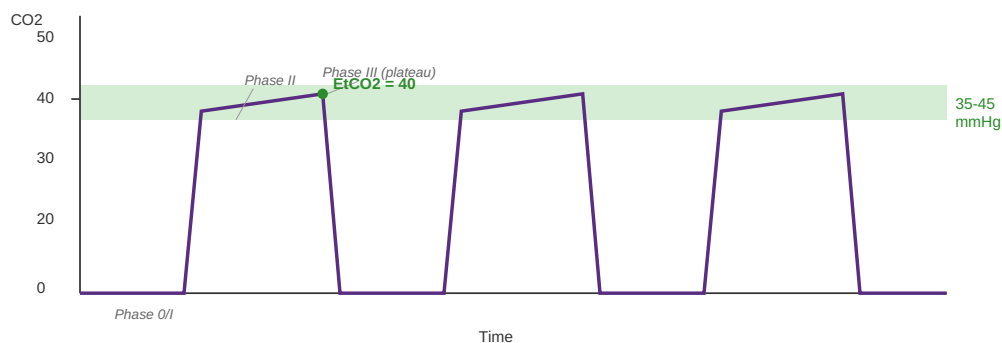


Figure 1.1 Normal capnogram showing the four phases of exhaled CO_2 . The end-tidal value is read at the very end of phase III, just before inspiration resumes. The normal EtCO_2 range of 35 to 45 mmHg is shown as the green band.

EtCO_2 is the most common bedside tool for monitoring ventilation during noninvasive positive pressure ventilation (NPPV) titration. A rising EtCO_2 alerts the technologist to worsening hypoventilation, and a drop of the trace to zero immediately identifies an apnea. The chief limitations are inaccuracy when the mask leaks (because exhaled gas escapes before reaching the sensor) and unreliable readings at very high respiratory rates. For these reasons EtCO_2 is usually complemented by TcCO_2 and SpO_2 during overnight studies.

Transcutaneous CO_2 (TcCO_2)

Transcutaneous CO_2 monitoring uses a small electrode applied to the skin (usually on the chest or forearm) and heated to 42 to 44 degrees Celsius. The heat dilates the capillaries directly under the electrode, and CO_2 from those capillaries diffuses up through the skin into a small chamber where it is measured. Because there is a small distance between the actual artery and the sensor, TcCO_2 typically reads 2 to 8 mmHg higher than the true arterial CO_2 , and the sensor must be calibrated before each study.

The major advantage of TcCO_2 is that it gives a continuous non-invasive estimate of CO_2 throughout the night, without being affected by mask leak. The main disadvantage is a 5 to 20 minute lag time before the displayed number catches up to a real change in PaCO_2 , which makes the modality best suited for trending across the night rather than for detecting fast changes. It is the modality of choice for overnight monitoring in obesity

hypoventilation syndrome (OHS), neuromuscular disease (NMD), congenital central hypoventilation syndrome (CCHS), the related disorder ROHHAD (described in Chapter 2), and almost all pediatric titrations.

Pulse Oximetry (SpO_2)

Pulse oximetry uses two wavelengths of light, red at 660 nanometers and infrared at 940 nanometers, shone through a fingertip or earlobe. Oxygenated hemoglobin (oxyhemoglobin) absorbs the two wavelengths differently from deoxygenated hemoglobin, and the ratio between the two absorptions yields the percentage of hemoglobin that is carrying oxygen. This principle is sometimes called the Beer-Lambert law.

A normal awake SpO_2 reading is 95 percent or higher. Readings below 90 percent are clinically concerning, and readings below 85 percent are critical. During sleep studies, the 4 percent oxygen desaturation index (the number of times per hour the SpO_2 drops by 4 percentage points or more) correlates well with the apnea-hypopnea index. Another useful summary number is the T90, which is the total time the SpO_2 is below 90 percent; it reflects the cumulative burden of nocturnal hypoxemia.

Pulse oximetry has several important limitations. Carbon monoxide poisoning produces carboxyhemoglobin, which absorbs light in a way that fools the sensor into reading nearly 100 percent regardless of the true oxygen content. Methemoglobinemia (an abnormal form of hemoglobin) causes the sensor to read approximately 85 percent no matter what the true saturation is. Motion, dark nail polish, poor peripheral perfusion, and severe anemia can all cause unreliable readings.

Arterial Blood Gas (ABG) Analysis

An arterial blood gas analysis directly measures the pH, the partial pressures of carbon dioxide and oxygen, the bicarbonate level, and the base excess in arterial blood. It is the gold standard against which the non-invasive monitors are compared. Normal values appear in the box below, followed by a summary table of the major acid-base disorders and their expected compensations.

Arterial Blood Gas - Normal Values

pH 7.35 to 7.45 | PaCO_2 35 to 45 mmHg | PaO_2 80 to 100 mmHg | HCO_3^- 22 to 26 mEq/L | SaO_2 at least 95 percent | Base excess plus or minus 2

Acid-Base Disorder	pH	PaCO ₂	HCO ₃ ⁻	Compensation Rule
Respiratory acidosis (acute)	Low	High	Normal	Bicarbonate rises 1 mEq/L for every 10 mmHg rise in CO ₂
Respiratory acidosis (chronic)	Low-normal	High	High	Bicarbonate rises 3.5 to 4 mEq/L for every 10 mmHg rise in CO ₂
Respiratory alkalosis (acute)	High	Low	Normal	Bicarbonate falls 2 mEq/L for every 10 mmHg fall in CO ₂
Respiratory alkalosis (chronic)	High-normal	Low	Low	Bicarbonate falls 4 to 5 mEq/L for every 10 mmHg fall in CO ₂
Metabolic acidosis	Low	Low (compensatory)	Low	Winter formula: expected PaCO ₂ = 1.5 × HCO ₃ + 8 (± 2)
Metabolic alkalosis	High	High (compensatory)	High	PaCO ₂ rises 0.7 mmHg for every 1 mEq/L rise in HCO ₃

Five-Step ABG Interpretation Method

Step 1: Look at the pH. A value below 7.35 is acidosis; above 7.45 is alkalosis.

Step 2: Look at the PaCO₂. Above 45 mmHg is a respiratory acidosis; below 35 mmHg is a respiratory alkalosis.

Step 3: Look at the bicarbonate. Below 22 mEq/L is a metabolic acidosis; above 26 mEq/L is a metabolic alkalosis.

Step 4: Match the primary disorder to the direction the pH actually moved.

Step 5: Check whether the compensation matches what the rules predict. If it does not, the patient probably has a mixed acid-base disorder.

Bonus calculation: the A-a gradient on room air (P_AO₂ minus PaO₂) is normally less than 15 mmHg. A widened gradient points to V/Q mismatch, true shunt, or a diffusion problem.

Chapter 2

Pathophysiology and Clinical Presentation - Domain 2 - Exam Weight 30 percent

2.1 Obstructive Sleep Apnea (OSA)

Adult OSA

Obstructive sleep apnea is a disorder of repetitive pharyngeal collapse during sleep despite continued ventilatory effort. The key events recognized on a polysomnogram are apneas (a complete cessation of airflow lasting 10 seconds or longer), hypopneas (a 30 percent or greater reduction in airflow accompanied by either a 4 percent oxygen desaturation or an arousal), and respiratory effort related arousals (abbreviated RERAs), which are subtle increases in respiratory effort that end in an arousal but do not meet the criteria for an apnea or hypopnea (6).

The underlying mechanism is reduced neuromuscular tone in the upper airway during sleep, which allows the soft palate or the base of the tongue (or both) to collapse against the posterior pharyngeal wall. The problem is made worse by obesity, the supine sleep position, rapid eye movement (REM) sleep, alcohol, sedatives, and certain craniofacial features such as a recessed lower jaw.

Risk Factors and Why They Matter

Several risk factors strongly predict OSA, and each has a physiologic reason behind it, which is worth understanding rather than just memorizing.

Risk Factor	Why It Increases OSA Risk
Male sex	Men have longer pharyngeal airways and more fat deposition around the upper airway than women, which makes the airway more prone to collapse.
Obesity (BMI > 30)	Fat deposits in and around the pharyngeal wall narrow the lumen, while abdominal fat reduces lung volume and removes caudal traction on the trachea, making the upper airway softer and more collapsible.
Age over 40	The pharyngeal dilator muscles lose tone and the upper airway tissues lose elasticity with age, both of which favor collapse.
Large neck circumference (> 17 in men, > 16 in women)	A large neck reflects fat accumulation around the upper airway and predicts a smaller pharyngeal lumen during sleep.
Retrognathia (a recessed lower jaw)	When the mandible sits posteriorly, the tongue base sits posteriorly too, which crowds the retroglossal airway.
Tonsillar hypertrophy	Enlarged tonsils take up space inside the oropharynx, narrowing the airway. This is the dominant cause in children.
Menopause	Premenopausal women are partly protected from OSA by two female hormones. Progesterone is a respiratory stimulant that increases upper-airway dilator muscle activity, and estrogen helps maintain muscle tone in the pharynx. After menopause, both hormones fall, the protective effect is lost, and fat is redistributed centrally to the neck and abdomen, all of which raise the risk of OSA to levels comparable to those seen in men.

AASM Severity Thresholds for Adult OSA

Apnea-Hypopnea Index (events/hour)	Severity	Clinical Note
5 to 14.9	Mild	Requires daytime symptoms (such as sleepiness) for diagnosis at this level.
15 to 29.9	Moderate	Diagnosis can be made on the AHI alone, regardless of symptoms.
30 or greater	Severe	High cardiovascular risk.
Per ICSD-3-TR (2) and the AASM 2017 diagnostic guideline (1), adult OSA is diagnosed when the obstructive RDI $\geq$ 5 events/h is accompanied by typical symptoms (sleepiness, unrefreshing sleep, fatigue or insomnia, awakening with gasping or choking, loud snoring, or witnessed apneas) OR when the obstructive RDI $\geq$ 15 events/h regardless of symptoms. Note that RDI as defined in ICSD-3 includes RERAs in addition to apneas and hypopneas; in clinical practice AHI and RDI are often used interchangeably, but the formal ICSD-3 criterion uses RDI	Diagnostic threshold	AASM 2017 adult diagnostic criteria (1).

The medical consequences of untreated adult OSA are substantial. Hypertension is present in 30 to 83 percent of patients with OSA, and the disorder is also strongly associated with coronary artery disease, atrial fibrillation, stroke, heart failure, type 2 diabetes mellitus, gastroesophageal reflux, pulmonary hypertension, and motor vehicle accidents from drowsy driving (1).

Pediatric OSA

The ICSD-3 and AASM diagnostic threshold for pediatric OSA is an obstructive AHI ≥ 1 event per hour, or a pattern of obstructive hypoventilation defined as hypercapnia ($\text{PaCO}_2 > 50$ mmHg) during $\geq 25\%$ of total sleep time accompanied by snoring, flattening of the nasal pressure waveform, or paradoxical thoracoabdominal motion (2,6). Some pediatric researchers and clinical guidelines use AHI ≥ 1.5 as a threshold for clinically significant pediatric OSA, but the AASM/ICSD-3 diagnostic criterion is ≥ 1 .

Children with OSA often do not appear sleepy in the daytime. Instead they tend to snore loudly, show paradoxical breathing (in which the chest sucks inward while the abdomen pushes outward during inspiration), wet the bed (enuresis), breathe through the mouth, sleep restlessly, and have behavior problems such as hyperactivity or poor school performance that can be mistaken for attention deficit hyperactivity disorder.

The first-line treatment for pediatric OSA is adenotonsillectomy (commonly written T and A) in children aged 2 to 18 years with adenotonsillar hypertrophy. If surgery is not curative, CPAP is the next step. A repeat polysomnogram is recommended 6 to 8 weeks after surgery to assess for residual OSA, which persists in approximately 25 to 40 percent of children, especially those who were obese before surgery.

Infant OSA and Special Considerations

Infants are anatomically different from older children. Their airway lumen is smaller in absolute terms, their ribs sit horizontally rather than obliquely (which reduces tidal volume), their arousal mechanisms are immature, and they may have specific vulnerabilities such as laryngomalacia (a floppy larynx) or micrognathia (a small jaw, as in Pierre Robin sequence). They are at risk for apparent life-threatening events, more recently renamed brief resolved unexplained events (ALTE or BRUE). Management includes cardiorespiratory monitoring, polysomnography, and either high-flow nasal cannula or nasal CPAP as first-line respiratory support.

2.2 Central Sleep Apnea (CSA)

Central sleep apnea occurs when the brainstem fails to send the drive signal to breathe. In contrast to obstructive events, where the patient is trying to breathe but cannot move air past a collapsed upper airway, in CSA the patient is not trying to breathe at all. This distinction is critical and is identified on the polysomnogram by the respiratory effort channels (chest and abdominal belts using respiratory inductance plethysmography, or RIP). During a central apnea, airflow ceases AND chest and abdominal effort go flat together. During an obstructive apnea, airflow ceases but chest and abdominal effort continue (and often grow more vigorous as the patient struggles against the closed airway). This effort comparison is illustrated in Chapter 5.

Note on subtype count. The *International Classification of Sleep Disorders, 3rd edition Text Revision (ICSD-3-TR, 2023)* (2) lists **six adult CSA categories**: primary CSA, CSA with Cheyne-Stokes breathing, CSA due to high altitude periodic breathing, CSA due to a medication or substance, treatment-emergent CSA, and CSA due to a medical disorder without Cheyne-Stokes. If primary CSA of infancy and primary CSA of prematurity are included, the total reaches eight. The subtypes are summarized in the sections that follow.

Primary (Idiopathic) Central Sleep Apnea

Primary CSA is diagnosed when no underlying cause (such as heart failure, opioid use, or high altitude) can be identified. The underlying mechanism is a low apneic carbon dioxide (CO_2) threshold. Every person has a CO_2 level below which the brainstem stops sending the breathing signal. In healthy individuals, this apneic

threshold sits comfortably below the normal sleeping PaCO_2 , so they never reach it. In primary CSA, the threshold is set unusually close to the resting PaCO_2 , so a small hyperventilation episode can drop the CO_2 below threshold and trigger an apnea. The pattern is most prominent during non-rapid-eye-movement (NREM) sleep stages N1 and N2, when ventilation depends most heavily on chemical drive. Treatment options include CPAP, acetazolamide (which produces a mild metabolic acidosis and raises the resting CO_2 away from the apneic threshold), adaptive servo-ventilation (ASV), and supplemental oxygen (5).

Cheyne-Stokes Breathing

Cheyne-Stokes breathing is a crescendo-decrescendo pattern of tidal volume that builds, peaks, then fades into a central apnea, and then repeats. The cycle typically lasts 45 to 90 seconds. The underlying mechanism is prolonged circulation time between the lungs and the central chemoreceptors in the medulla, which is common in congestive heart failure (CHF), stroke, and renal failure. Because the chemoreceptors receive delayed information about lung CO_2 , they over-respond and then over-correct, producing the oscillation. Treatment is to optimize the underlying condition first (heart failure management is most important), then consider supplemental oxygen, acetazolamide, or ASV when the left ventricular ejection fraction (LVEF) is preserved (above 45 percent) (5).

CRITICAL EXAM POINT - ASV Contraindication (SERVE-HF Trial, NEJM 2015) (3)

Adaptive servo-ventilation is **CONTRAINDICATED** when **ALL THREE** of the following are present simultaneously:

- Symptomatic congestive heart failure (NYHA class II to IV)
- Left ventricular ejection fraction (LVEF) at or below 45 percent
- Predominantly central sleep apnea

Why? The SERVE-HF trial (3) showed a 34 percent relative increase in cardiovascular mortality (hazard ratio 1.34; 95% CI 1.09 to 1.65; $p = 0.006$) in this specific patient population when treated with ASV. The mechanism is not fully understood but may involve suppression of compensatory hyperventilation that the failing heart relies upon. If LVEF is above 45 percent, ASV remains appropriate. Always document the most recent echocardiogram before initiating ASV.

CLINICAL PEARL - CSA Treatment Sequence (Guideline-First, Not Device-First)

A common exam trap is to jump straight to ASV for any patient with central sleep apnea. The published guideline sequence (5) is usually:

Step 1. Optimize the underlying medical condition first. For CSA with heart failure, this means guideline-directed medical therapy (GDMT) for CHF - ACE inhibitors or ARBs/ARNIs, beta-blockers, mineralocorticoid antagonists, SGLT2 inhibitors, and diuretics as appropriate. Effective CHF treatment alone often substantially reduces CSA.

Step 2. Try CPAP first. CPAP is appropriate initial PAP therapy for CSA with CHF - it improves cardiac function, reduces afterload, and resolves CSA in a significant proportion of patients (the CANPAP study population (4)).

Step 3. Add supplemental oxygen if hypoxemia persists or for high-altitude or idiopathic CSA. The AASM conditionally suggests low-flow oxygen for CSA due to heart failure and CSA due to high altitude (5).

Step 4. If CPAP is not tolerated or fails, the next step depends on LVEF.

- **Preserved EF (> 45 percent):** ASV (with supplemental oxygen if needed) is the preferred advanced therapy.
- **Reduced EF ($\leq$ 45 percent) with symptomatic CHF:** Historically (SERVE-HF, 2015) (3), ASV was considered CONTRAINDICATED in this group. Use BPAP-ST or transvenous phrenic nerve stimulation (remedé) instead.

Exam phrasing matters: "initial treatment," "first-line," and "most appropriate first" all point to the earliest guideline step (medical optimization or CPAP), NOT the most advanced device.

2025 AASM CSA Guideline Update - Reinstatement of ASV

In late 2025, the AASM published an updated clinical practice guideline for treatment of CSA in adults (Badr et al., *J Clin Sleep Med*, 2025) (5). This guideline conditionally suggests ASV for CSA across multiple etiologies, including primary CSA, CSA due to heart failure, CSA due to medication or substance use, treatment-emergent CSA, and CSA due to a medical condition.

Important caveats:

- ASV use in heart failure with reduced ejection fraction (HFrEF) should be limited to experienced centers with close monitoring and follow-up.
- Increased mortality in the SERVE-HF trial was attributed to one specific device algorithm; safety data are not available for all ASV devices.
- The guideline suggests AGAINST BPAP without a backup rate, because it may worsen central apneas.
- CPAP remains a suggested option for most CSA etiologies as initial therapy.

Exam relevance: The BRPT ATC examination may still test the historical SERVE-HF framework (ASV contraindicated when CHF + LVEF $\leq$ 45% + predominantly CSA) because exam content lags behind clinical guidelines. Know BOTH frameworks. When the exam stem describes a patient meeting the SERVE-HF triad, the expected answer is to AVOID ASV.

High Altitude Periodic Breathing

At high altitude (typically above 2,500 meters), low atmospheric oxygen triggers hyperventilation, which drives the PaCO₂ below the apneic threshold and produces central apneas in a short cycle of about 12 to 34 seconds (cycle length shortens as altitude increases). The pattern is normal in healthy individuals during the first few nights at altitude and usually resolves with acclimatization. Acetazolamide is highly effective both for prevention and treatment, and supplemental oxygen or descent to lower altitude will also resolve the pattern.

Treatment-Emergent Central Sleep Apnea (TECSA)

Treatment-emergent CSA, also called complex sleep apnea, refers to central apneas that appear or persist after CPAP is initiated for what was diagnosed as obstructive sleep apnea. At baseline the patient had obstructive events, so the diagnosis was OSA, but once CPAP eliminates the obstructions, central apneas emerge or fail to resolve. The mechanism is that CPAP eliminates the hypercapnic arousals that previously kept the patient's CO₂ above the apneic threshold; with those arousals gone, the CO₂ falls and central apneas unmask. Management is to continue CPAP, because in approximately 80 percent of cases the central apneas resolve spontaneously within 4 to 12 weeks. If they persist, ASV is the most effective treatment (provided the LVEF is preserved) (5). Always rule out opioid use, which is the most common reversible cause.

Medication- and Substance-Induced CSA

Opioids are the most common medication cause of central sleep apnea. They suppress the pre-Bötzinger complex (the central pattern generator in the medulla that produces the breathing rhythm) and blunt both the hypercapnic and hypoxic ventilatory responses. Methadone carries the highest risk because of its long half-life and active metabolites; long-acting formulations carry more risk than short-acting ones. The typical pattern is

ataxic breathing (also called Biot's breathing) with irregular rate and depth. Benzodiazepines and Z-drugs (zolpidem, eszopiclone) also blunt HCVR and HVR, and combined use with opioids is synergistic and dangerous. Management starts with dose reduction of the offending agent whenever feasible. When PAP is needed, bilevel PAP in spontaneous-timed mode (BPAP-ST) with a backup rate is appropriate, and ASV may be used if the LVEF is above 45 percent (5).

Apnea of Prematurity

Apnea of prematurity is defined as an apneic pause longer than 20 seconds, or a shorter pause accompanied by bradycardia or desaturation, in an infant born before 37 weeks gestational age. The underlying problem is immaturity of the central respiratory control system. First-line treatment is caffeine citrate, a methylxanthine that stimulates the central nervous system and improves diaphragmatic contractility. Theophylline is a second-line methylxanthine, and doxapram is reserved as a last resort. Nasal CPAP or HFNC supports the airway when needed. Apnea of prematurity typically resolves by 36 to 44 weeks post-menstrual age.

CSA Due to a Medical Condition

Condition	Mechanism and Clinical Notes
Chronic Kidney Disease / End-Stage Renal Disease	Metabolic acidosis triggers compensatory hyperventilation, which lowers CO ₂ below the apneic threshold. Fluid overload also worsens upper airway edema. Pattern often improves with effective dialysis.
Brainstem injury or tumor	Direct disruption of the medullary rhythm generators. Pattern may be ataxic (Biot's) or apneustic. Mechanical ventilation is often required.
Multiple System Atrophy	Combination of vocal cord paresis with central respiratory dysfunction. Nocturnal stridor is characteristic. CPAP is generally contraindicated; tracheostomy is often needed.
Parkinson's Disease	Upper-airway rigidity, REM sleep behavior disorder, and a mix of obstructive and central events. CPAP is first-line.
Arnold-Chiari Malformation	Brainstem compression by cerebellar tonsillar herniation produces central apneas. Surgical decompression may resolve the pattern; BPAP may bridge the patient until surgery.

2.3 Hypoventilation Syndromes

Hypoventilation is diagnosed when the awake arterial CO₂ (PaCO₂) exceeds 45 mmHg, or when nocturnal SpO₂ falls below 90 percent for at least 5 minutes in the setting of sleep-related hypercapnia (6). The primary problem is inadequate breathing depth or rate, not upper airway obstruction. This is an important distinction, because hypoventilation cannot be treated by CPAP alone; the patient needs pressure support (a bilevel device) or a volume-targeted mode to actively augment tidal volume.

Obesity Hypoventilation Syndrome (OHS)

Obesity hypoventilation syndrome is defined by three criteria, all of which must be present: a body mass index (BMI) of 30 or higher, awake PaCO_2 above 45 mmHg, and absence of any other explanation for the hypoventilation (7). Approximately 10 to 20 percent of obese patients with OSA also have OHS, and roughly 90 percent of patients with OHS also have coexisting OSA.

The mechanism involves several layers. The mechanical load of chest wall and abdominal adiposity reduces functional residual capacity and increases the work of breathing. Leptin resistance (leptin normally stimulates the respiratory center) further blunts the hypercapnic ventilatory response. As CO_2 retention becomes chronic, the kidneys retain bicarbonate (HCO_3^-) to compensate, which normalizes pH but also further blunts the central chemoreceptor drive. The cycle becomes self-reinforcing.

Screening Pearl - Serum Bicarbonate

A serum bicarbonate of 27 mEq/L or higher is a useful screening marker for OHS (7). The kidneys retain bicarbonate to compensate for chronic CO_2 retention, so an elevated HCO_3^- in an obese patient with OSA should prompt confirmatory arterial blood gas testing.

Clinical features include severe excessive daytime sleepiness (EDS), polycythemia (the body produces more red blood cells in response to chronic hypoxemia), pulmonary hypertension, and right heart failure (cor pulmonale). First-line treatment is BPAP or AVAPS (average volume-assured pressure support) when hypoventilation is the dominant problem (7). CPAP alone may suffice if the OSA component is what is driving the hypoxemia, but most patients with established OHS need active ventilatory assistance. Weight loss is essential for durable improvement, and bariatric surgery produces the most reliable long-term resolution.

CLINICAL PEARL - OHS Hypoventilation Starts in REM Sleep

On the polysomnogram, OHS hypoventilation appears **FIRST** and is **WORST** in REM sleep, then extends into NREM, and eventually becomes a daytime problem.

Why? REM sleep produces near-total skeletal muscle atonia, including the accessory respiratory muscles (intercostals, sternocleidomastoid, scalenes). The diaphragm is preserved during REM, but in an obese patient the diaphragm alone cannot overcome the mechanical load of chest-wall and abdominal adiposity. Tidal volume falls, minute ventilation drops, and CO_2 climbs. Hypoxemia in OHS therefore typically begins as profound REM-related desaturation before any daytime ABG abnormality is detectable.

Clinical implications: nocturnal oximetry plus transcutaneous CO_2 (TcCO_2) monitoring can detect OHS years before a resting daytime PaCO_2 exceeds 45 mmHg. REM-period hypoventilation is also why the same physiology drives early hypoventilation in neuromuscular disease.

Congenital Central Hypoventilation Syndrome (CCHS)

Congenital central hypoventilation syndrome (CCHS), historically called Ondine's curse, is a rare genetic disorder caused by mutations in the PHOX2B gene (8). The mutation is most commonly a polyalanine repeat

expansion, the inheritance pattern is autosomal dominant, and the majority of cases arise from new (de novo) mutations rather than being inherited from a parent. Longer polyalanine repeat expansions produce more severe disease.

The hallmark of CCHS is a complete absence of both the hypercapnic ventilatory response (HCVR) and the hypoxic ventilatory response (HVR). The patient simply does not sense rising CO₂ or falling oxygen, has no sensation of dyspnea, and will hypoventilate to dangerous levels without external warning. Hypoventilation is worst during NREM sleep, when chemical drive is normally most important. Autonomic dysregulation is also common (abnormal pupillary responses, esophageal dysmotility, arrhythmias). **Approximately 20 percent** of CCHS patients (most commonly in the polyalanine repeat mutation, or PARM, subgroup) have Hirschsprung disease; rates are substantially higher in the rarer non-PARM (NPARM) subgroup. There is also an elevated risk of neural crest tumors such as neuroblastoma.

Treatment is lifelong ventilatory support during sleep, and in severe cases continuously. Options include tracheostomy with mechanical ventilation (severe forms), BPAP during sleep (milder forms), and phrenic nerve pacing (8). There is no pharmacologic cure.

ROHHAD Syndrome

ROHHAD stands for **R**apid-onset **O**besity with **H**ypothalamic dysfunction, **H**ypoventilation, and **A**utonomic **D**ysregulation. It presents between ages 1.5 and 7 years with rapid weight gain as the first sign, followed months to years later by hypoventilation (9). ROHHAD is distinguished from CCHS by being PHOX2B negative, by its later onset, by the fact that obesity and hypothalamic dysfunction precede hypoventilation, and by the fact that approximately 40 percent of affected children have associated neural crest tumors. Management requires lifelong ventilatory support and surveillance for tumors.

Medication- and Substance-Induced Hypoventilation

Opioids suppress both the rate and depth of breathing. Methadone and fentanyl patches carry the highest risk because of prolonged duration of action. The combination of opioids with benzodiazepines or gabapentinoids is synergistic and substantially increases the risk of fatal hypoventilation. Management requires minimization of the offending agents whenever clinically feasible. BPAP with a backup rate is the preferred PAP mode. Supplemental oxygen alone does NOT correct hypoventilation; in fact, in CO₂ retainers, high-flow oxygen can worsen hypercapnia by abolishing the hypoxic drive.

Neuromuscular Disease and Restrictive Chest Wall

Neuromuscular diseases (NMDs) impair the respiratory pump. The lungs themselves may be normal, but the muscles cannot generate adequate ventilation, and as the disease progresses the patient develops nocturnal hypoventilation first, then daytime hypoventilation, then respiratory failure. Nocturnal NIV (noninvasive ventilation) prolongs survival and improves quality of life in most of these disorders.

Disorder	Features	NIV Threshold and Notes
Amyotrophic Lateral Sclerosis (ALS)	Mixed upper and lower motor neuron disease; bulbar involvement is common and worsens secretions; progressive course.	Forced vital capacity (FVC) every 3 months. Start NIV when FVC drops below 50 percent of predicted or when symptoms appear. In the Bourke 2006 randomized trial (10), NIV extended median survival by approximately 7 months, with greater benefit in patients with non-bulbar onset and little or no survival benefit in those with severe bulbar dysfunction.
Duchenne Muscular Dystrophy	X-linked recessive; respiratory failure is the leading cause of death.	Start NIV when FVC drops below 50 percent or with nocturnal desaturation. Cough assist device is essential. Mechanical ventilation will eventually be required.
Spinal Muscular Atrophy (SMA)	Anterior horn cell disease; type I is most severe (infantile onset).	Early initiation of NIV; cough assist is mandatory; new genetic therapies (nusinersen, onasemnogene) are improving outcomes.
Myasthenia Gravis	Neuromuscular junction autoimmune disease with fluctuating weakness.	Myasthenic crisis is a medical emergency. Intubate if FVC drops below 1 liter or is rapidly declining.
Guillain-Barré Syndrome	Acute ascending demyelinating polyneuropathy; 25 to 30 percent of patients eventually need ventilation.	Intubate when FVC falls below 20 mL/kg or MIP drops below 30 cmH ₂ O (the rule of 20-30).
Kyphoscoliosis	Severe spinal curvature restricts chest expansion; Cobb angle above 100 degrees confers high respiratory failure risk.	Early BPAP prevents acute exacerbations and reduces hospitalizations.

NMD Respiratory Monitoring Thresholds

FVC below 50 percent predicted: initiate NIV.

Maximum Inspiratory Pressure (MIP) below 60 cmH₂O: clinically significant inspiratory muscle weakness.

Peak Cough Flow (PCF) below 160 L/min: cough is ineffective; mechanical cough assist is mandatory.

PCF below 270 L/min: cough becomes inadequate during upper respiratory infections; prophylactic cough assist is recommended.

Sniff Nasal Inspiratory Pressure (SNIP) below 40 cmH₂O: indicates significant weakness, especially useful in bulbar disease where MIP measurement is unreliable.

Nocturnal oximetry plus TcCO₂: detect nocturnal hypoventilation before daytime ABG values change.

Chapter 3

Evaluation and Treatment - Domain 3 - Exam Weight 50 percent

Domain 3 represents half of the BRPT Advanced Titration Certificate examination (18), and rightly so: the practical knowledge required to safely and effectively titrate positive airway pressure therapy is the core of what an advanced sleep technologist does. This chapter walks through the official AASM titration guidelines, then covers each PAP modality in detail, followed by oxygen therapy, oral appliances, neurostimulators, and other treatment options.

3.1 Official AASM PAP Titration Guidelines (Kushida 2008)

The AASM Clinical Guidelines for the Manual Titration of Positive Airway Pressure (Kushida et al., *J Clin Sleep Med* 2008) (11) remain the foundational document for adult PAP titration and are heavily tested on the BRPT examination. The full guideline runs more than 30 pages; what follows is a structured summary of the testable elements.

Pre-Titration Requirements

Before the patient arrives, several requirements must be met. A formal diagnosis of OSA must already be established by a previous polysomnogram or by a home sleep apnea test in an appropriate candidate (1); titration is not a diagnostic study. The patient must be educated about the purpose of CPAP and given a chance to try the mask before the study begins; many studies fail not because the pressure was wrong but because the patient could not tolerate the interface. Mask selection (nasal, nasal-pillows, oronasal) should be guided by patient anatomy, mouth breathing, and any prior tolerance issues.

Starting Pressure

Patient Population	Starting CPAP Pressure
Adults	4 cmH ₂ O (no lower)
Children at least 12 years old	4 cmH ₂ O
Children under 12	4 cmH ₂ O
Maximum recommended CPAP pressure	20 cmH ₂ O in adults; 15 cmH ₂ O in children. If higher pressures are needed, switch to bilevel.

When to Increase the Pressure

The pressure should be raised when respiratory events recur. The Kushida guideline (11) specifies a minimum number of events to be observed at a given pressure before increasing, so that the technologist is not chasing isolated events that would resolve on their own. The criteria are:

Event Type	Number Required to Justify a Pressure Increase
Obstructive apneas	At least 2
Hypopneas	At least 3
Respiratory effort related arousals (RERAs)	At least 5
Loud or unambiguous snoring	At least 3 minutes

Pressure Increment and Interval

Pressure should be raised in increments of 1 cmH₂O, and the minimum interval between any two increases is 5 minutes (11). The technologist should not go above 20 cmH₂O on a CPAP titration; if events persist at 15 cmH₂O or pressure intolerance develops, the appropriate response is to switch to bilevel rather than to continue raising the single pressure.

Bilevel PAP Titration

When switching from CPAP to BPAP, EPAP is set to the CPAP pressure that last eliminated obstructive events, or to 4 cmH₂O if starting fresh. IPAP is then set 4 cmH₂O above EPAP to give a starting pressure support (PS) of 4 cmH₂O. The minimum PS is 4 cmH₂O; below this the device functions as CPAP and provides no inspiratory augmentation. The maximum recommended IPAP is 30 cmH₂O in adults and 20 cmH₂O in children (11). Increases should be applied to whichever pressure addresses the residual problem: increase IPAP and EPAP for residual obstructive events, increase IPAP for residual hypopneas or RERAs.

Titration Outcome Grading

The Kushida guideline (11) grades the result of the titration into four categories. These grades are heavily tested on the BRPT examination.

Grade	Definition
Optimal	RDI is reduced to less than 5 events per hour at the selected pressure during at least 15 minutes of recording, AND must include supine REM sleep at that pressure not continually interrupted by spontaneous arousals.
Good	RDI is reduced to 10 or less, OR by at least 50 percent if baseline RDI is less than 15, AND must include supine REM sleep at the selected pressure not continually interrupted by spontaneous arousals.
Adequate	Either: (1) does not reduce RDI to 10 or less but achieves a 75 percent reduction from baseline (especially in severe OSA); OR (2) meets the criteria for "Good" except that supine REM sleep did not occur at the selected pressure.
Unacceptable	Does not meet any of the above grades. A repeat titration study should be considered.

† **Note on RDI in this section:** Kushida 2008 defines **RDI = apneas + hypopneas + RERAs per hour of sleep** and explicitly states the term is **NOT** synonymous with the **AHI** (which counts only apneas and hypopneas). When applying the

grading criteria above on the BRPT exam, use RDI as defined by the guideline. This distinction is a common source of confusion because modern clinical practice often uses RDI and AHI interchangeably.

REM Rebound and Supine REM

REM rebound, the increased proportion of REM sleep that follows a period of REM suppression, is common in newly treated OSA patients and tends to occur in the second half of the night. Supine REM is the worst-case scenario for an OSA patient because the gravitational pull on the tongue and soft palate is greatest in the supine position and upper-airway dilator tone is lowest during REM. The AASM specifically requires that an optimal titration demonstrate control of breathing during supine REM (11), because if the patient is controlled in supine REM, almost any other body position and sleep stage will also be controlled.

3.2 PAP Devices - Clinical Reference

Six PAP modalities are in routine clinical use. Each has a specific physiology, indications, contraindications, and titration approach. The waveform for each modality is shown in the figure that accompanies its section, and a consolidated comparison table appears at the end of section 3.2.

CPAP - Continuous Positive Airway Pressure

CPAP delivers a single, fixed pressure throughout the entire respiratory cycle. The mechanism is a pneumatic splint: the constant intraluminal pressure keeps the upper airway open and prevents the pharyngeal collapse that defines OSA. CPAP does not actively assist ventilation; the patient breathes spontaneously against a fixed back-pressure. It is the first-line therapy for adult OSA. Pressure range is typically 4 to 20 cmH₂O.

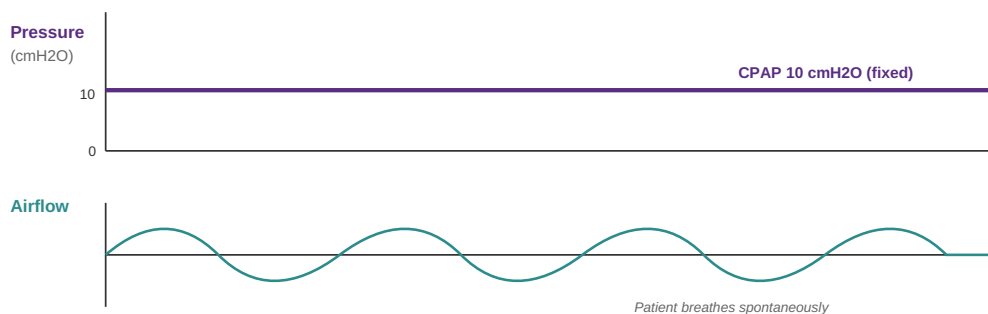


Figure 3.1 CPAP delivers a single fixed pressure throughout the entire breath cycle. Airflow (lower trace) reflects the patient's spontaneous breathing against the fixed pneumatic splint.

APAP - Auto-titrating Positive Airway Pressure

APAP devices automatically adjust pressure within a clinician-set range (commonly 5 to 15 cmH₂O) based on real-time detection of flow limitation, snoring, and apneas. The pressure can be reported as a 95th percentile, which is the pressure at or below which the device operated for 95 percent of the recorded time. APAP is appropriate for uncomplicated moderate-to-severe adult OSA without significant comorbidities. **APAP is NOT appropriate** for CSA, OHS, Cheyne-Stokes breathing, neuromuscular disease, or significant cardiovascular disease, because the auto-titrating algorithm assumes obstructive physiology and may respond inappropriately to central events.

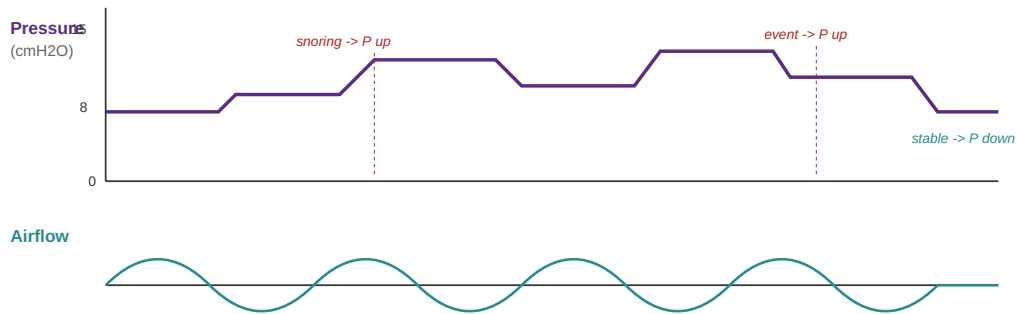


Figure 3.2 APAP pressure rises in response to events such as snoring or flow limitation, then reduces when the airway is stable. Set within a clinician-determined min-max range.

BPAP - Bilevel Positive Airway Pressure

BPAP delivers two distinct pressure levels: a higher inspiratory positive airway pressure (IPAP) during patient-triggered inspiration, and a lower expiratory positive airway pressure (EPAP) during expiration. The difference between them is the pressure support (PS), which actively augments the patient's tidal volume. EPAP eliminates obstructive events (it functions as the pneumatic splint, exactly like CPAP); IPAP eliminates hypopneas and RERAs (it actively helps the patient inhale). Indications include CPAP intolerance at high pressures, hypoventilation, neuromuscular weakness, and CO₂ retention. Minimum PS is 4 cmH₂O; below that the device is functionally CPAP (11).

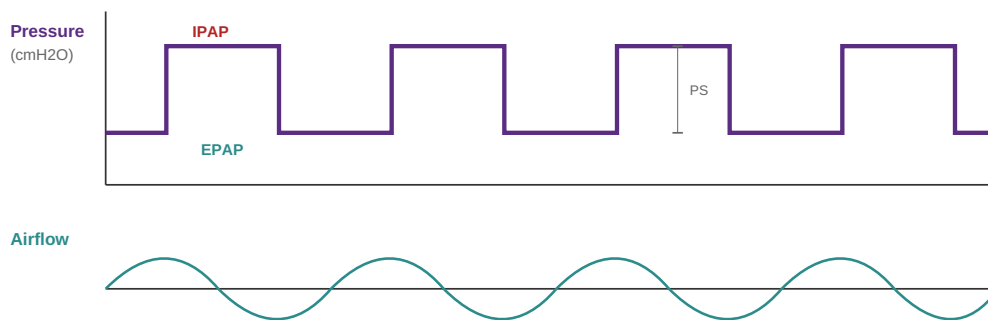


Figure 3.3 BPAP cycles between IPAP and EPAP synchronously with the patient's breath. PS = IPAP - EPAP.

BPAP-ST - Spontaneous/Timed Mode

BPAP-ST adds a backup respiratory rate to the spontaneous bilevel mode. The patient still triggers most breaths (the spontaneous part), but if the patient fails to take a breath within the clinician-set time interval, the machine delivers a timed mandatory breath at the IPAP pressure (the timed part). This makes BPAP-ST the preferred mode for any patient at risk of central apnea or frank apnea, including OHS with central events, opioid-induced CSA, neuromuscular disease, severe COPD, and CCHS. The backup rate is typically set 2 breaths per minute below the patient's spontaneous awake rate.

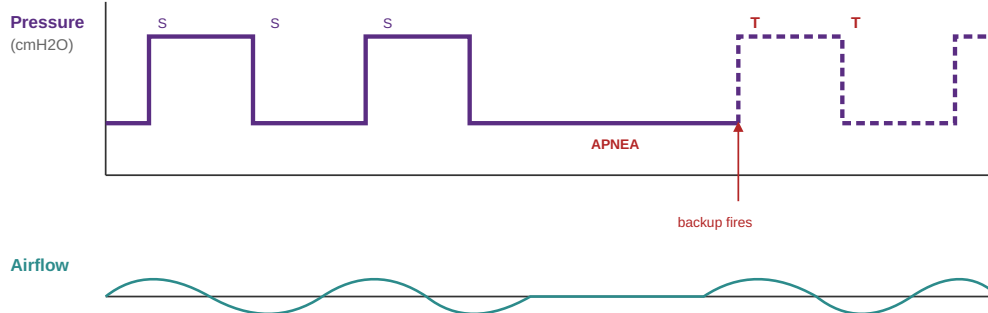


Figure 3.4 BPAP-ST: patient triggers most breaths (S); if an apnea exceeds the backup interval, the machine fires a timed breath (T, shown dashed).

ASV - Adaptive Servo-Ventilation

Adaptive servo-ventilation continuously monitors the patient's minute ventilation and adjusts pressure support breath-by-breath to target 90 to 95 percent of the recent rolling average. The result is active suppression of Cheyne-Stokes oscillations and other forms of central or complex sleep apnea. ASV is the preferred therapy for Cheyne-Stokes with preserved LVEF, treatment-emergent CSA that fails to resolve on CPAP, and complex (mixed) sleep apnea. The 2025 AASM CSA guideline (5) expanded its conditional indications across multiple etiologies.

REPEAT WARNING - ASV CONTRAINDICATION

ASV is contraindicated when ALL THREE of the following are present: (1) symptomatic CHF, (2) LVEF at or below 45 percent, and (3) predominantly central sleep apnea. The SERVE-HF trial (3) demonstrated a **34 percent relative increase** in cardiovascular mortality (HR 1.34; 95% CI 1.09 to 1.65; $p = 0.006$) in this population.

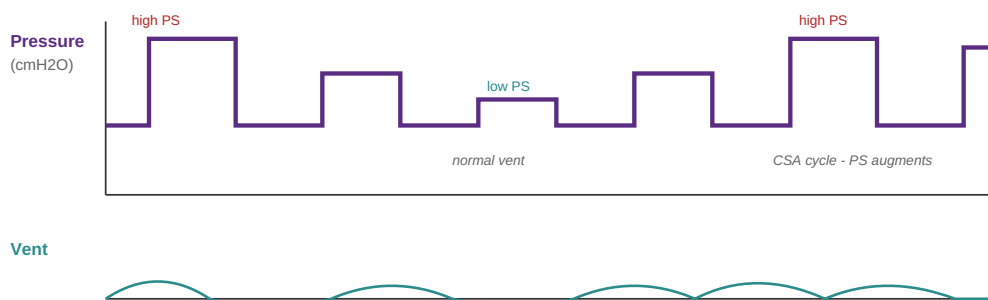


Figure 3.5 ASV pressure support varies dynamically to counter Cheyne-Stokes cycling and maintain stable minute ventilation.

AVAPS - Average Volume-Assured Pressure Support

AVAPS is a hybrid mode that combines bilevel pressure delivery with a clinician-set target tidal volume. The device monitors the actual tidal volume the patient is achieving and adjusts IPAP automatically, breath-over-breath, to keep the volume at the target (typically 8 to 10 mL per kilogram of ideal body weight). This is particularly useful when lung mechanics or muscle strength vary across the night, as in OHS (7), advanced

neuromuscular disease, and CCHS (8). Adjustments are slow and stepwise to avoid abrupt pressure changes that would disrupt sleep.

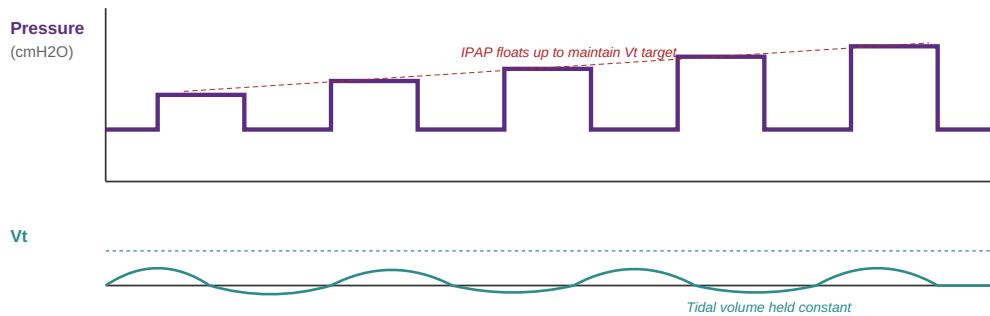


Figure 3.6 AVAPS: IPAP floats breath-over-breath to keep tidal volume at the clinician-set target.

PAP Mode Comparison Table

Mode	Pressure Pattern	First-Line For	Notes
CPAP	Single fixed pressure	Adult OSA	No active ventilation
APAP	Auto-adjusting within range	Uncomplicated OSA	Avoid in CSA, OHS, NMD, CHF
BPAP	IPAP + EPAP, no backup	CPAP intolerance at high pressure, hypoventilation	Minimum PS 4 cmH ₂ O
BPAP-ST	IPAP + EPAP + backup rate	OHS, CSA, CCHS, NMD	Backup typically 2 breaths below spontaneous awake rate
ASV	Variable PS auto-adjusting	Cheyne-Stokes (preserved EF), TECSA	CONTRAINDICATED: CHF + LVEF≤45 + CSA
AVAPS	Variable IPAP to target Vt	OHS, advanced NMD, CCHS	Vt = 8 to 10 mL/kg IBW

3.3 Oxygen Therapy

Supplemental oxygen plays a defined but limited role in sleep medicine. It corrects hypoxemia but does not treat the underlying ventilatory abnormality and is therefore rarely used as a sole therapy for sleep-disordered breathing. The principal indications are nocturnal hypoxemia from interstitial lung disease, residual hypoxemia on optimized PAP therapy, central sleep apnea (adjunctive) (5), and high-altitude periodic breathing.

Delivery Device	Flow Range or Setting	Estimated FiO ₂	Clinical Note
Nasal cannula	1 to 6 L/min	24 to 44 percent	Comfortable; most common method for sleep lab use
Simple face mask	5 to 10 L/min	40 to 60 percent	Minimum 5 L/min flow to prevent CO ₂ rebreathing
Non-rebreather mask	10 to 15 L/min	60 to 95 percent	Reservoir bag must remain inflated
Venturi mask	Color-coded ports	24 to 50 percent precise	Best for CO ₂ retainers needing precise FiO ₂
High-flow nasal cannula	Up to 60 L/min	Up to 100 percent	Heated, humidified; provides modest PEEP effect

3.4 Oral Appliance Therapy

Oral appliances are removable dental devices worn during sleep to reposition the lower jaw forward (mandibular advancement devices, or MADs) or to hold the tongue forward (tongue-retaining devices, or TRDs). By advancing the mandible 5 to 10 millimeters, the tongue base is pulled forward and the retroglossal airway is enlarged, reducing OSA severity. Oral appliances are fitted by qualified dentists with training in dental sleep medicine, not by sleep technologists, but the technologist should understand their place in the treatment algorithm.

Indication or Limitation	Note
First-line for	Mild-to-moderate OSA; primary snoring
Second-line for	CPAP-intolerant moderate-to-severe OSA
NOT for	Severe OSA (less effective than CPAP); edentulous patients; active TMJ disorder
Common side effects	Tooth movement, TMJ discomfort, excess salivation, dry mouth
Follow-up	PSG with the appliance in place to verify treatment efficacy

3.5 Neurostimulator Therapy

Three neurostimulator devices are currently in use for sleep-related breathing disorders. Each targets a different physiologic problem and has different indications. The technologist may not directly manage these devices, but understanding their place in the treatment algorithm is heavily tested on the BRPT exam.

Hypoglossal Nerve Stimulator (Inspire®)

The hypoglossal nerve stimulator (HNS) is an implanted device that delivers electrical stimulation to the hypoglossal nerve (cranial nerve XII), which controls the tongue. Synchronizing the stimulation with the

patient's inspiration causes the genioglossus to contract and the tongue to protrude, opening the retroglossal airway during the most vulnerable phase of breathing. The patient activates the device with a handheld remote at bedtime.

Selection Criteria	Note
Age	The primary FDA indication remains age ≥ 22 years (PMA P130008, 2014)
AHI	15 to 100 events per hour per current FDA indication, expanded from the original 15 to 65 range in June 2023 (P130008/S090). Some BRPT exam materials may still reference the original 15 to 65 range
BMI	At or below 40 kg/m ² per current FDA indication, expanded from the prior ≤ 32 in June 2023 (P130008/S090). The STAR trial enrolled patients with BMI 32 or below; some BRPT exam materials may still reference the older threshold
Anatomy	DISE (drug-induced sleep endoscopy) must show absence of complete concentric collapse at the soft palate
Central event burden	Central and mixed apneas must comprise less than 25 percent of the total AHI
PAP history	Documented CPAP intolerance or failure of an adequate trial
Trial outcome	STAR trial (Strollo, NEJM 2014) (12): in the responder analysis at 12 months, 83 of 126 patients (66 percent) were classified as responders by the primary efficacy criteria. Median AHI fell from 29.3 to 9.0 events per hour, and median ODI fell from 25.4 to 7.4.

Phrenic Nerve Stimulator (remedé®)

The remedé system is a transvenously implanted device that stimulates the phrenic nerve, causing diaphragmatic contraction and producing a breath. It is used for moderate-to-severe central sleep apnea in adults. Unlike the hypoglossal nerve stimulator, the remedé device activates automatically at preset bedtime hours.

Indication or Trial Datum	Detail
Indication	Moderate-to-severe CSA in adults
Mechanism	Transvenous electrode at the phrenic nerve; the device delivers stimulation during preset sleep hours
Pivotal trial	Costanzo et al., <i>Lancet</i> 2016 (13) (randomized controlled trial, 6-month primary endpoint)
Primary endpoint	51 percent of treatment patients (35 of 68) achieved at least 50 percent AHI reduction at 6 months, compared with 11 percent of controls (8 of 73). The between-group difference was 41 percentage points (95 percent CI, 25 to 54; $p < 0.0001$). Controls had the device implanted but inactive (i.e., not a sham), and the study was not blinded to the investigator.
Adverse events	Lead displacement; local site infection; need for system revision in a minority

Daytime Tongue Stimulator (eXciteOSA®)

eXciteOSA is the first FDA-cleared awake (daytime) neuromuscular electrical stimulator for the treatment of mild OSA and primary snoring. The patient holds the mouthpiece against the tongue once daily during waking hours; treatment is intended to improve genioglossus muscle tone over time. The device received FDA De Novo clearance (DEN200018) in 2021 (14).

Item	Detail
Indication	Mild OSA (AHI less than 15) and primary snoring in adults at least 18 years old
Use	20 minutes once daily during wakefulness for an initial 6-week treatment phase, followed by once-weekly maintenance per the FDA-cleared regimen (14). Some clinical protocols use a more frequent maintenance schedule, but the FDA-cleared labeling specifies once weekly.
Mechanism	Awake daytime neuromuscular electrical stimulation of intrinsic and extrinsic tongue muscles to improve genioglossus tone
FDA-listed adverse events	FDA-listed adverse events Excessive salivation, tongue or tooth discomfort, tongue tingling, dental filling sensitivity, metallic taste, gagging, and tight jaw (per FDA De Novo decision summary DEN200018; the agency did not rank these events by frequency) (14)
NOT for	Moderate-to-severe OSA; patients with cardiac pacemakers; intraoral metal implants

3.6 Other Treatment Options

High-Flow Nasal Cannula (HFNC)

High-flow nasal cannula delivers heated, humidified air (with or without supplemental oxygen) through a wide-bore nasal interface at flow rates up to 60 liters per minute. The high flow flushes anatomic dead space, generates a modest amount of positive end-expiratory pressure (PEEP) in the pharynx (approximately 2 to 5 cmH₂O), and provides excellent humidification, which improves tolerance for patients who would not accept a sealed mask interface. HFNC has a defined role in infant and pediatric OSA, in adults with mild OSA who refuse CPAP, and as a bridge to PAP therapy in acute respiratory failure.

Surgical Treatment

Surgical options for OSA include uvulopalatopharyngoplasty (UPPP), maxillomandibular advancement (MMA), genioglossus advancement, midline glossectomy, and adenotonsillectomy (the dominant procedure in children). MMA is the most effective adult surgical procedure with success rates as high as 75 to 90 percent, but it is a major surgery and is reserved for selected patients.

Behavioral and Lifestyle Interventions

Behavioral interventions include weight loss (a 10 percent weight reduction lowers AHI by approximately 25 to 30 percent), positional therapy for patients with exclusively or predominantly supine OSA, avoidance of alcohol within 3 to 4 hours of bedtime, avoidance of sedative hypnotics, smoking cessation, and treatment of nasal congestion. These are not substitutes for PAP therapy in severe OSA but they are useful adjuncts and may be sufficient as monotherapy in mild positional OSA.

Chapter 4

Snoring and Pediatric PAP Titration

4.1 Snoring: Evaluation and Treatment

What Causes Snoring

Snoring is the sound produced by vibration of the soft tissues of the upper airway, principally the soft palate and uvula, as turbulent air flows past them. It indicates partial narrowing of the airway, even when no apneas or hypopneas are scoreable on the polysomnogram. Primary (simple) snoring is defined by an apnea-hypopnea index below 5 events per hour and absence of any clinically significant excessive daytime sleepiness; if either of those conditions is exceeded, the diagnosis is OSA rather than primary snoring.

Upper Airway Resistance Syndrome (UARS)

Upper airway resistance syndrome sits between primary snoring and frank OSA. The patient has snoring plus EEG arousals triggered by increased respiratory effort (RERAs), even though the AHI may be below 5. Patients are often non-obese, often female, and frequently present with insomnia, fatigue, and unrefreshing sleep rather than the classic OSA picture of loud snoring with witnessed apneas. Treatment is essentially identical to mild OSA: CPAP, mandibular advancement device, or positional therapy depending on severity, with attention to coexisting nasal pathology.

Treatment of Primary Snoring

Intervention	Indication and Comment
Weight loss	Reduces upper-airway adipose loading; first-line where applicable.
Positional therapy	For patients whose snoring is dominantly supine.
Alcohol avoidance	Alcohol increases pharyngeal relaxation and worsens snoring; avoid within 3 to 4 hours of bedtime.
Nasal interventions	Treat allergic rhinitis, septal deviation, and turbinate hypertrophy when contributing.
Mandibular advancement device	Effective for primary snoring as well as mild OSA.
eXciteOSA®	FDA-cleared for primary snoring and mild OSA, daytime tongue stimulation, 20 minutes/day (14).
Surgical (palatal)	UPPP, palatal stiffening procedures; reserved for select cases with anatomic indication.

4.2 Pediatric PAP Titration

The AASM Clinical Guideline for the Manual Titration of Positive Airway Pressure (Kushida et al., 2008) (11) contains a dedicated pediatric section. Pediatric titration differs from adult titration in several important ways: the event-scoring criteria are different, the maximum pressures are lower, and the technologist must work much harder on behavioral preparation before the study night.

Pediatric Respiratory Event Criteria

Event	Pediatric Definition
Obstructive apnea	At least 2 missed breaths or at least 90 percent reduction in airflow lasting the duration of 2 breaths, with continued respiratory effort
Central apnea	Absent airflow AND absent respiratory effort lasting at least 20 seconds, OR shorter event if accompanied by a 3 percent oxygen desaturation, an arousal, or bradycardia
Hypopnea	At least 30 percent airflow reduction for at least 2 breaths PLUS either a 3 percent oxygen desaturation or an arousal
Diagnostic OSA threshold	Obstructive AHI of 1 or greater

Pediatric Titration Parameters (11)

Parameter	Children Under 12 Years Old	Children 12 Years and Older
CPAP starting pressure	4 cmH ₂ O	4 cmH ₂ O
CPAP maximum pressure	15 cmH ₂ O	20 cmH ₂ O
BPAP IPAP maximum	20 cmH ₂ O	30 cmH ₂ O
Events to trigger pressure increase	1 obstructive apnea, OR 1 hypopnea, OR 3 RERAs, OR at least 1 minute of loud snoring	2 obstructive apneas, OR 3 hypopneas, OR 5 RERAs, OR at least 3 minutes of loud snoring
Minimum interval between increases	5 minutes	5 minutes
Pressure increment	1 cmH ₂ O	1 cmH ₂ O

Pediatric Behavioral Preparation

Behavioral acclimation is the single most important determinant of long-term pediatric PAP adherence. Recommended steps include desensitization sessions before the titration night, mask choice by the child when

possible, the presence of a parent or caregiver during the night study, reward systems and reinforcement, and a child life specialist for children with developmental disabilities or anxiety.

CLINICAL PEARL - Pediatric Titration Pitfalls

- The pediatric event-scoring thresholds are MORE sensitive than the adult thresholds (lower limits trigger an event sooner). Do not apply adult rules to a pediatric study.
- The pediatric pressure cap is LOWER than the adult cap (CPAP ≤ 15 , BPAP IPAP ≤ 20 cmH₂O).
- The pressure-increase event threshold is LOWER (a single obstructive apnea is enough in children under 12).
- Mask fit is critical: many "treatment failures" in pediatric clinic are mask problems, not pressure problems.
- Use TcCO₂ monitoring routinely; pulse oximetry alone misses early hypoventilation in children.

Chapter 5

Illustrated Waveform Atlas

This chapter is a visual reference. Every breathing pattern, every effort-channel signature, and every PAP-mode waveform that appears on the BRPT examination is summarized below. Each figure includes a short explanatory caption; the diagnostic and treatment context for each pattern is in the chapters indicated.

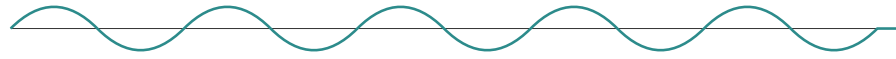
5.1 Breathing Pattern Waveforms

The composite figure below shows the six classic breathing patterns described in Chapter 1.3, arranged for side-by-side comparison. Pay particular attention to the rate, the depth, and the regularity of each pattern; the underlying lesion or condition that produces each pattern is also given. The central sleep apnea panel additionally shows the corresponding effort channel, which goes flat together with airflow; contrast this with the obstructive sleep apnea pattern in Figure 5.2.

Six Classic Breathing Patterns

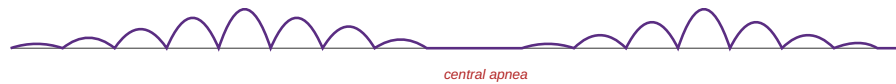
A. Eupnea (normal)

Regular rate and depth, 12-20/min



B. Cheyne-Stokes

Crescendo-decrescendo with central apnea (45-90 s cycle)



C. Biot's (ataxic)

Irregular rate and depth, unpredictable apneas (medullary lesion)



D. Apneustic

Prolonged inspiratory hold, brief expiration (pontine lesion)



E. Kussmaul

Deep, rapid, sighing breaths (metabolic acidosis)



F. Central Sleep Apnea

Airflow absent AND effort absent (compare to OSA, next figure)

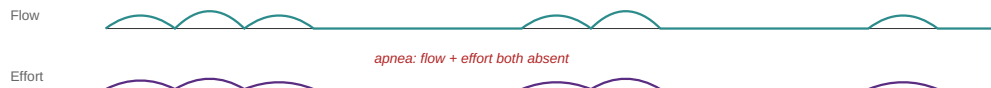


Figure 5.1 Composite of the six breathing patterns encountered in clinical sleep medicine. A: eupnea (normal). B: Cheyne-Stokes (crescendo-decrescendo with central apnea, 45-90 second cycle; CHF, stroke, renal failure). C: Biot's ataxic breathing (irregular rate AND depth; medullary lesion). D: apneustic (prolonged inspiratory hold; pontine lesion). E: Kussmaul (deep, rapid, sighing; metabolic acidosis). F: central sleep apnea (airflow AND effort both flat). See Chapter 1.3 for mechanism and Chapter 2.2 for management.

5.2 Central vs Obstructive Apnea - Effort Channel Comparison

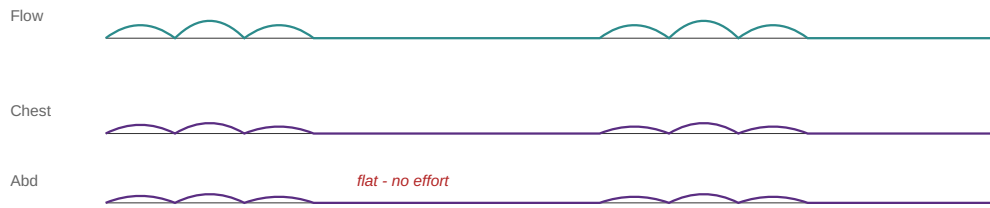
Distinguishing central from obstructive apneas on the polysomnogram is a high-yield exam topic. The single most reliable discriminator is the respiratory effort channel (chest and abdominal bands, measured by respiratory inductance plethysmography). In a central apnea, airflow ceases AND effort goes flat together, because the brainstem is not sending the drive signal. In an obstructive apnea, airflow ceases but effort

continues - and often intensifies, with paradoxical motion of the chest and abdomen, as the patient struggles against the closed upper airway.

Central vs Obstructive Apnea

Central Sleep Apnea (CSA)

No drive signal -> airflow AND effort both go flat



Obstructive Sleep Apnea (OSA)

Airway closed -> airflow STOPS but effort CONTINUES (often intensifies)

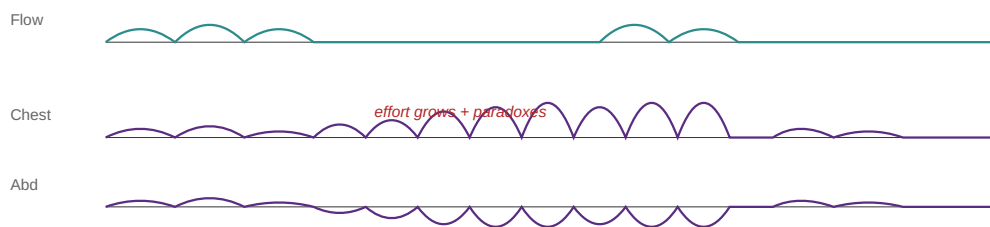


Figure 5.2 Top: central sleep apnea. Airflow stops AND effort (chest and abdomen) goes flat together. Bottom: obstructive sleep apnea. Airflow stops, but chest and abdominal effort continue and often paradox against each other as the patient struggles against the closed airway.

5.3 PAP Mode Waveforms

The six waveforms below summarize the pressure (and where relevant, the volume) delivery patterns of each PAP modality. Use these alongside the device descriptions in Chapter 3.2.

CPAP - Single Fixed Pressure

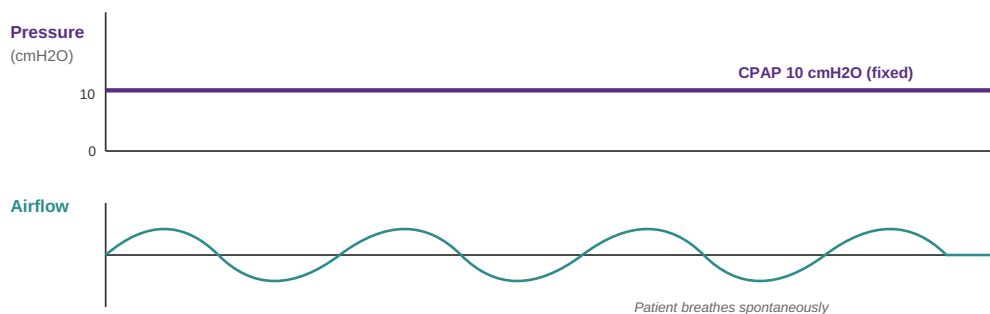


Figure 5.3 CPAP delivers a single fixed pressure (here 10 cmH₂O) throughout the entire respiratory cycle. The airflow trace below reflects the patient's spontaneous breathing against the fixed pneumatic splint.

APAP - Auto-Adjusting Pressure

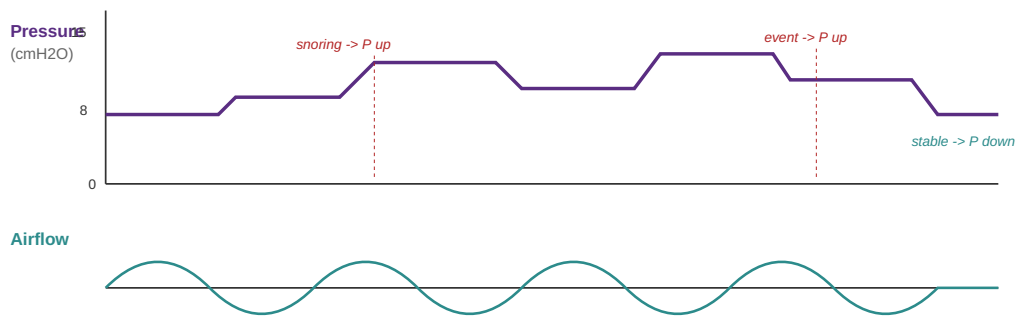


Figure 5.4 APAP pressure rises in response to flow limitation or snoring and falls when the airway is stable, all within a clinician-set min-max range. Inappropriate for CSA, OHS, CHF, or NMD.

BPAP - Bilevel

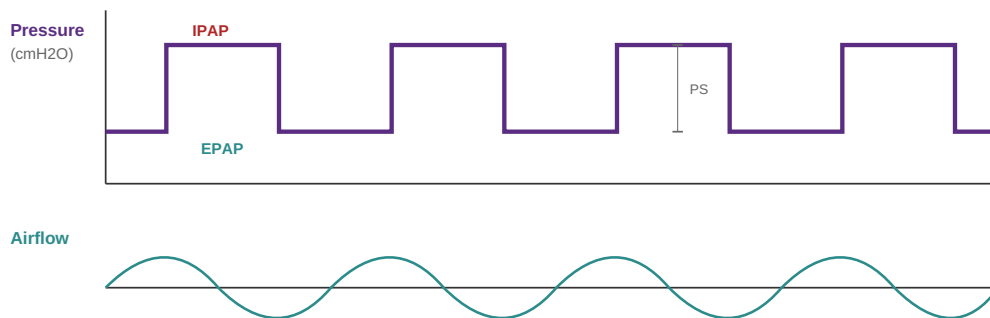


Figure 5.5 BPAP alternates between IPAP (during inspiration) and EPAP (during expiration). The difference between them is the pressure support (PS), which actively augments tidal volume.

BPAP-ST - Spontaneous/Timed

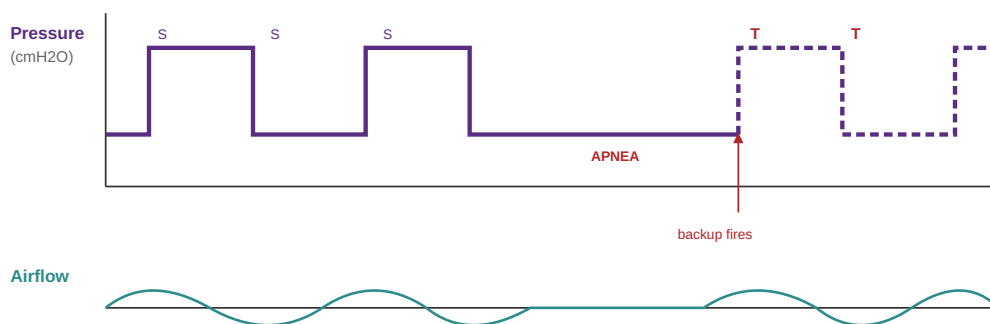


Figure 5.6 BPAP-ST behaves like BPAP for spontaneous breaths (S). If an apnea exceeds the backup interval, the device delivers a timed mandatory breath (T, shown dashed). Indicated for OHS, opioid-induced CSA, neuromuscular disease, and CCHS.

ASV - Adaptive Servo-Ventilation

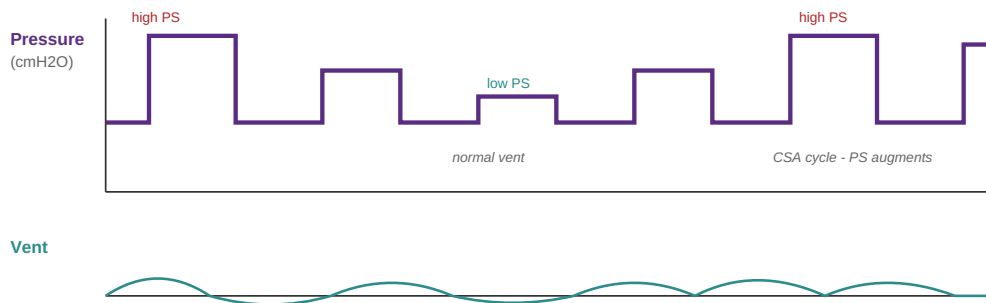


Figure 5.7 ASV continuously adjusts pressure support breath-by-breath to suppress Cheyne-Stokes oscillation and target stable minute ventilation. Contraindicated when CHF + LVEF $\leq$ 45 percent + predominantly CSA are all present (SERVE-HF) (3).

AVAPS - Average Volume-Assured Pressure Support

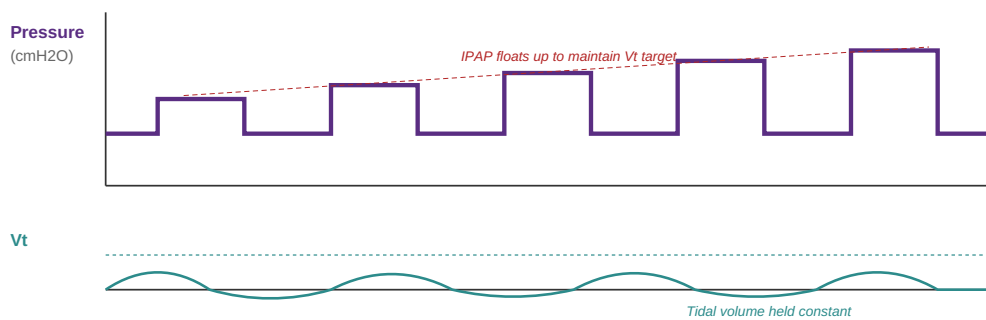


Figure 5.8 AVAPS adjusts IPAP gradually breath-over-breath to maintain a clinician-set target tidal volume (here held constant on the lower trace). Useful for OHS, advanced NMD, and CCHS where lung mechanics vary across the night.

Chapter 6

Quick Reference - Key Numbers and Exam Summary

This chapter consolidates the highest-yield numbers, thresholds, and rules from the preceding chapters. It is designed to be reviewed in the final week before the BRPT Advanced Titration Certificate examination, when the goal is recall rather than learning. Use it alongside the Chapter 5 waveform atlas.

6.1 Diagnostic Thresholds

Disorder	Diagnostic Criterion	Source
Adult OSA	AHI ≥ 5 with symptoms, OR AHI ≥ 15 regardless of symptoms	AASM 2017 (1)
Pediatric OSA	Pediatric OSA Obstructive AHI ≥ 1 (ICSD-3/AASM diagnostic threshold) AASM Scoring Manual (6), ICSD-3-TR (2)	AASM Scoring Manual (6)
Hypoventilation (adult)	Awake PaCO ₂ > 45 mmHg, or nocturnal SpO ₂ < 90% for ≥ 5 min	AASM Scoring Manual (6)
OHS	BMI ≥ 30 + awake PaCO ₂ > 45 + no other cause	ATS 2019 (7)
HCO ₃ ⁻ screening threshold for OHS	HCO ₃ ⁻ ≥ 27 mEq/L should prompt ABG	ATS 2019 (7)
Adult OSA severity	Mild 5-14.9; Moderate 15-29.9; Severe ≥ 30	AASM (1)

6.2 PAP Titration Quick Reference (Kushida 2008) (11)

Parameter	Adult	Pediatric (< 12 y)
Starting CPAP	4 cmH ₂ O	4 cmH ₂ O
Max CPAP	20 cmH ₂ O	15 cmH ₂ O
Max BPAP IPAP	30 cmH ₂ O	20 cmH ₂ O
Increment	1 cmH ₂ O	1 cmH ₂ O
Interval	≥ 5 min between increases	≥ 5 min between increases
Events to trigger increase	≥ 2 obstructive apneas, ≥ 3 hypopneas, ≥ 5 RERAs, ≥ 3 min snoring	≥ 1 apnea, ≥ 1 hypopnea, ≥ 3 RERAs, ≥ 1 min snoring
Minimum BPAP PS	4 cmH ₂ O	4 cmH ₂ O

6.3 Titration Outcome Grades (RDI)

Grade	RDI Criterion
Optimal	RDI < 5, with supine REM at the selected pressure not continually interrupted by spontaneous arousals
Good	RDI ≤ 10, OR reduced by ≥ 50% if baseline < 15, with supine REM at the selected pressure
Adequate	Either (1) does not reach RDI ≤ 10 but achieves ≥ 75% reduction, OR (2) meets "Good" except supine REM did not occur
Unacceptable	Does not meet any of the above. Repeat titration recommended.

6.4 ABG Quick Reference

Value	Normal Range
pH	7.35 - 7.45
PaCO ₂	35 - 45 mmHg
PaO ₂	80 - 100 mmHg
HCO ₃ ⁻	22 - 26 mEq/L
SaO ₂	≥ 95%
A-a gradient (room air)	< 15 mmHg
EtCO ₂	35 - 45 mmHg (2-5 mmHg below PaCO ₂)

6.5 Critical Contraindications and Warnings

ASV - SERVE-HF Contraindication (3)

ASV is CONTRAINDICATED when ALL THREE apply: symptomatic CHF (NYHA II-IV), LVEF ≤ 45%, predominantly central sleep apnea. The 2025 AASM CSA guideline (5) conditionally permits ASV in HFrEF only at experienced centers with close monitoring.

Oxygen Caution in CO₂ Retainers

High-flow oxygen can abolish the hypoxic drive in CO₂ retainers (severe COPD, OHS, CCHS) and worsen hypercapnia. Use a Venturi mask with a target SpO₂ of 88-92 percent.

Neurostimulator Eligibility

Hypoglossal nerve stimulator (Inspire) requires DISE showing absence of complete concentric collapse at the soft palate, and central + mixed apneas comprising < 25% of total AHI (12). Current FDA indication: age ≥22 for the primary indication (with separate expanded indications for ages 18 – 21 since April 2020 and ages 13 – 18 with Down syndrome since March 2023); AHI 15 – 100 and BMI ≤40 since June 2023.

Phrenic nerve stimulator (remedé) is for moderate-to-severe CSA (13); eXciteOSA is for mild OSA and primary snoring only (14).

6.6 Exam Strategy - High-Yield Patterns and Common Traps

The ten patterns below describe the most common ways the BRPT examination probes the same underlying concept. Recognize the pattern, and the answer becomes much easier.

1. **"Most appropriate first step" usually means a guideline-mandated low-cost step.** If the stem describes a patient with newly diagnosed CSA and CHF, the first step is medical optimization of CHF, then CPAP, NOT an immediate jump to ASV (5).
2. **"Contraindicated" is a flag for SERVE-HF triad.** The combination of CHF + reduced EF + predominantly CSA in the stem strongly suggests the answer involves avoiding ASV (3).
3. **REM-related desaturation in an obese patient = OHS.** The combination of profound REM desaturation, an obese BMI, and elevated HCO_3^- on chemistry panel should lead you to OHS rather than pure OSA (7).
4. **Pediatric criteria differ from adult criteria.** Pediatric scoring uses 2 missed breaths for apnea duration, ≥1 AHI threshold, and lower pressure caps (11). Do not apply adult numbers to a pediatric scenario.
5. **Effort channel discriminates central from obstructive apnea.** When the question shows a tracing or describes one, look at the effort channel: flat = central; continued or paradoxical = obstructive.
6. **"Floppy infant with central apneas and Hirschsprung" = CCHS.** The PHOX2B genetic test is diagnostic.
7. **"Rapid weight gain after age 2 followed by central hypoventilation" = ROHHAD.** Distinguished from CCHS by being PHOX2B negative and later in onset.
8. **"Ataxic irregular breathing in a chronic-pain patient" = opioid-induced CSA.** First-line PAP modality is BPAP-ST with a backup rate.
9. **"Cycle length of 12-34 seconds at altitude" = high altitude periodic breathing.** Acetazolamide and oxygen are the first interventions.
10. **Watch for unit traps.** Pressure is cmH_2O , partial pressure is mmHg, oxygen flow is L/min, FiO_2 is a percentage. Confused units waste careful reasoning.

6.7 Seven-Day Review Plan

Day	Focus	Reference
Day 1	Re-read Chapter 1 (physiology and monitoring); memorize the alveolar gas equation and the ABG five-step method.	Ch 1
Day 2	OSA: severity thresholds, pediatric vs adult event criteria, treatment algorithm.	Ch 2.1
Day 3	CSA: six adult subtypes, SERVE-HF, CANPAP, 2025 AASM update (5).	Ch 2.2
Day 4	Hypoventilation: OHS, CCHS, ROHHAD, NMD; REM-period physiology.	Ch 2.3
Day 5	PAP modes and Kushida titration rules; oxygen therapy.	Ch 3.1-3.3
Day 6	Oral appliances, neurostimulators (Inspire, remedé, eXciteOSA), surgical/behavioral.	Ch 3.4-3.6
Day 7	Quick Reference (this chapter); waveform atlas (Chapter 5); 10 exam-strategy patterns above.	Ch 5, 6

Chapter 7

References

References are formatted in APA (7th edition) style and listed in the numeric sequence used throughout the text — that is, in order of first appearance. Each entry is numbered to match the in-text citation, allowing readers to follow any citation directly to its source. Where a single reference is cited at multiple points, the same number is reused without renumbering. Digital Object Identifiers (DOIs) are provided as resolvable https links; where no DOI exists (books, manuals, and agency publications), the publisher or a stable URL is given instead. A comprehensive alphabetical index follows this reference list.

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